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ABDOMINAL PAIN AND ITS ASSOCIATED REFLEX PHENOMENA.[1]

BEING AN INTERPRETATION OF ABDOMINAL PAIN ILLUSTRATED BY A SERIES OF CASES.

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I.—INTRODUCTORY.

As early as 1753 Haller failed to find evidence that ordinary stimuli applied to the pleura, peritoneum and abdominal viscera could cause pain. Considerably more than a century later it was found on further investigation that the stomach of man could be touched, cauterized or incised only after a subcutaneous injection of cocaine without pain resulting therefrom. The problem set as a result of these investigations has led to a series of accurate experiments, with a view to ascertaining why pain is felt as a result of the disturbance of apparently insensitive viscera. Lennander in an exhaustive series of experiments proved that no pain resulted from manipulations, such as palpation of the fundus of the gall-bladder, the application of artery forceps to its serous layer, cauterization, the application of silver nitrate and aspiration of the organ. Although these manipulations produced no sensation, he found that in the process of opening the abdominal cavity the division of the aponeuroses of the external oblique caused pain. However:

> After infiltration (with local anæsthetic) between the aponeuroses of the external oblique and between the *internus* and *transversus abdominis* muscles, these structures can be divided without expression of pain on the part of the patient. On division of a nerve in the abdominal wall a momentary pain was felt. In the area supplied by the divided nerve no pain was felt in the parietal serosa, either when incised or pinched, but outside this area severe pain was produced by the same stimuli. After infiltration of the extra-peritoneal areolar tissue, the parietal peritoneum became insensitive over the rather limited area efficiently charged by the fluid. In the other parts not reached by the anæsthetic the pain on manipulation of the parietal peritoneum was severe.

Sir James Mackenzie (1) who quotes the above statement, confirmed these observations. "The sensitive layer," he says, "is the layer of loose connective tissue lying immediately outside the peritoneum. I suspected its presence for a long time, as I could frequently get exquisite tenderness in pushing my fingers between the recti muscles, for instance, in the case of gastric ulcer, the stomach not being affected by the pressure." Ramstrom, as Mackenzie goes on to point out, has made a careful histological examination of the abdominal wall. He has shown that the layer mentioned is highly endowed with nerves and nerve endings. The fact, as Lennander showed, that there is an area of anæsthesia of the parietal serosa after a nerve of the abdominal wall has been divided, proves that the sensory nerves of the abdominal wall supply these fibres.

[1] Arguments and illustrative cases used for Sandes's Surgical Prize Essay for final degree candidates, 1920, reproduced by courtesy of the Department of Surgery, Sydney University.

This striking fact has attracted the attention of many observers. But it will readily be seen, as they have seen, that this single system cannot alone be concerned in the entire series of phenomena of abdominal pain, tenderness and rigidity, associated with abdominal disturbance. It must be borne in mind that a number of layers exists between the outer world and the lumen of the alimentary canal. These layers are represented clearly in Figure I.,

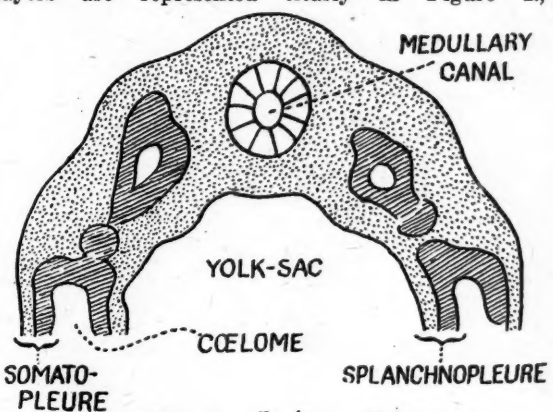

Fig.1. T.S. of an Embryo 2·5 m.m. long (McMurrich, after Van Lenhossek.)

where the somato-pleure and splanchno-pleure are shown separated by the peritoneal cavity. The outer layer later receives a sheet of voluntary muscle tissue and is clothed by ectoderm. The wall of the gut on the other hand consists of involuntary muscle lined by endoderm.

Bichât maintained, until Halier found the white *ramus communicans*, that these two great muscular systems were innervated by two separate nervous systems which had no connexion.

The purpose of this essay is to approach the problem from a clinical standpoint and to interpret abdominal pain and the reflex phenomena inseparable from it by the fullest consideration of the possible connexions between the somatic and splanchnic nervous systems.

The primary conception one has of the spinal cord in approaching any clinical problem is that it consists of the connexions of a series of outgoing and incoming fibres, together with the reflex connexions between these two sets. The afferent fibres form the posterior ganglionated nerve root and the efferent fibres form the anterior nerve root of a typical spinal nerve, the two roots combining to form the mixed nerve trunk (see Diagram). This trunk divides into anterior and posterior rami which supply the body wall. This is the somatic portion of the nerve. But in addition, the anterior nerve root is accompanied by sympathetic efferent nerve fibres, which leave the anterior ramus as the white *ramus communicans* and pass to the sympathetic ganglia connected with the

spinal nerve. This ganglion sends post-ganglionic fibres to various destinations. Afferent sympathetic fibres also pass to the cord in the posterior nerve root. There is thus indicated the well-defined division of a typical spinal nerve into a somatic element to and from the structures of the body wall and a splanchnic or visceral element to involuntary musculature and from mucous surfaces.

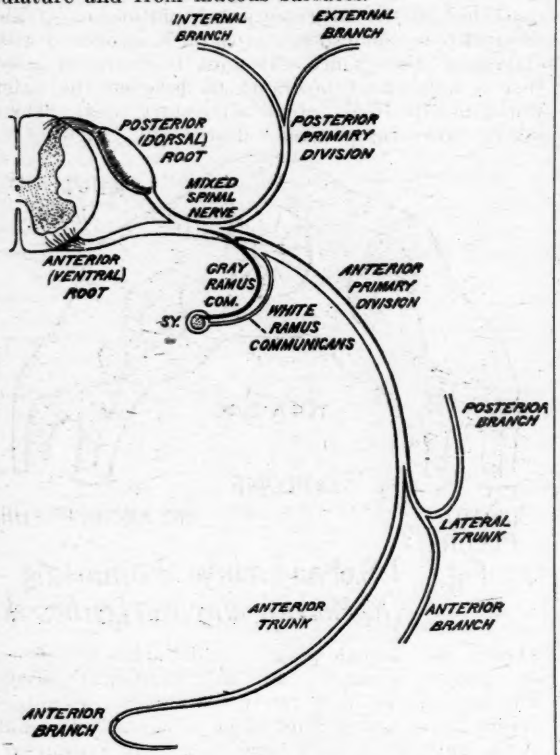

Diagram.

Each of these systems will now be considered separately in more detail, more especially in regard to the nervous connexions of the layers of tissue intervening between the external surface of the body and the interior of the alimentary canal.

A.—The Somatic Nervous System.

The abdominal wall is supplied by the anterior primary divisions or rami of spinal nerves, from the 7th thoracic to the 1st lumbar nerve. These rami may be localized by remembering that the 7th thoracic reaches the mid-line at the level of the xiphoid process, the 10th of the umbilicus and the 12th of the upper margin of the *symphysis pubis*. The elements of these branches which supply the general body wall—the somatic fibres—are functionally divisible into:

(*a*) Motor fibres.
(*b*) Sensory fibres.

(1) The motor fibres supply the voluntary musculature of the abdominal wall. They are the axones of nerve cells situated in the anterior horn of grey matter within the spinal cord, which pass through the anterior nerve root to the mixed nerve trunk and thence by the anterior rami to the abdominal muscles.

(2) The sensory fibres begin in terminals which may be situated in the skin, the extra-peritoneal fat or the muscles, the last named to transmit impulses of deep sensibility to the spinal cord.

These sensory nerves are the peripheral processes of nerve cells situated within the dorsal spinal ganglia. The central processes of these cells pass into the spinal cord by the posterior nerve root. The nerve endings (exteroceptive or proprioceptive receptors) of any of these sensory fibres will, on receiving appropriate stimuli, transmit pain impulses along the nerve fibres to the spinal cord. Here the impulses are conveyed by collaterals and terminals to the posterior horn of grey matter. The cells around which these branches aborize, I propose to designate the "pain group" of cells. The axones of these cells form the second relay of conduction. They cross to the opposite lateral funiculus and contribute towards the formation of the posterior (lateral) spinothalamic path of that side. This tract passes to the thalamus along with the medial lemniscus or fillet within the brain stem. Here synapsis occurs with a third nerve cell and the axone of this thalamic cell passes by way of the internal capsule to the cortex. In the thalamus or cortex or both, according to Head's recent work (11), the pain impulse is converted into a pain sensation and consciousness of pain results. I propose to designate the second and third relays of this tract the "central pain path."

B.—The Splanchnic Nervous System.

Under this heading all the nervous connexions of involuntary musculature and of the mucous membrane and solid organs derived from the splanchnopleure are included. As in the somatic system two sets of fibres can be distinguished:—

(1) Efferent splanchnic fibres.
(2) Afferent splanchnic fibres.

(1) The efferent or motor splanchnic fibres come from three sources:—

(*i.*) In the thoracic and lumbar regions of the spinal cord a group of small cells situated between the anterior and posterior horns of grey matter and forming the lateral column are seen more plainly than elsewhere. The axones of these cells are prolonged as fine medullated fibres. They pass out with the anterior nerve roots and leave the spinal nerve as the white *rami communicantes*. These white rami are present from the first thoracic to the third lumbar nerves. This has been designated the thoraco-lumbar outflow or true sympathetic nervous system. The white rami pass to the sympathetic ganglia of the ganglionated chain to the side and in front of the vertebral bodies. Here many branch around the sympathetic nerve cells. The majority of the axones of these cells are non-medullated. Many of these non-medullated fibres pass back to the spinal nerve *viâ* the grey rami which they constitute and are distributed with it to the sweat glands and the involuntary musculature of the arteries of the general abdominal wall.

Some fibres pass on beyond the ganglion through the ganglionated cord to reach *inter alia* the ganglia of the cervical region, whence grey rami (although no white rami are present) also pass to the cervical

nerves to be distributed to the glands and vascular muscle of the head and neck. Some fibres also pass on as splanchnic nerves to reach the entire alimentary canal, in order to form synapses with the ganglionic neurones in the involuntary muscle of its wall. These fibres are always pre-ganglionic and after passing through or over the vertebral ganglia of the ganglionated cord, they branch about ganglionated neurones either those of the prevertebral ganglia (*e.g.*, semilunar or coeliac), or those of the visceral ganglia. The latter occur, for instance, as the plexus of Auerbach, which is situated between the internal circular and the external longitudinal muscle coats.

On stimulation these sympathetic efferent fibres cause dilatation of the gut and flaccidity of its muscle layer.

(*ii.*) The second set of splanchnic efferents issues from the vagus nucleus. These fibres are also pre-ganglionic and pass into the plexus of Meissner, below the mucous membrane. These fibres when stimulated cause contraction of the gut wall. They pass distally as far as the caecum and perhaps as far as the left colic flexure.

(*iii.*) The contraction of the remaining portion of the gut wall is brought about by the pelvic nerves. These nerves are splanchnic efferent fibres which come from the second, third and fourth sacral areas and without communicating with the sympathetic ganglionated cord, pass to the gut wall.

These last two sets form the cranio-sacral outflow.

(2) The splanchnic afferent fibres accompany these three systems of efferent fibres.

(*i.*) There are sympathetic afferent fibres accompanying the splanchnic nerves back to the spinal cord by passing through the posterior nerve root. Hardesty (2) claims that these fibres have their cells either in the spinal ganglion or in the sympathetic ganglion.

(*ii.*) The vagus contains sensory fibres, the cells of which are in the *ganglion jugulare*; the peripheral processes pass centrally from the gut wall and the central processes end in the receptive nucleus of the vagus.

(*iii.*) Similar fibres with their cells in the spinal ganglia of the second, third and fourth sacral nerves doubtless form afferent paths of the pelvic nerves.

To summarize, it will be seen that a typical spinal nerve consists of two parts: somatic and splanchnic (or visceral). Each of these parts further contains afferent and efferent fibres. As Edinger (3) shows clearly (Figure II.) we must therefore recognize four great morphological elements in such a spinal nerve:

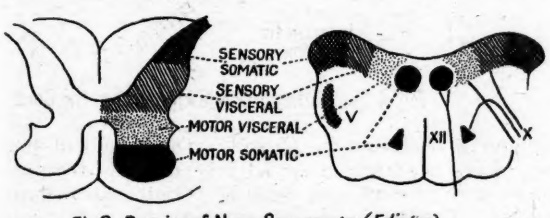

Fig. 2. Doctrine of Nerve Components (*Edinger*)

(1) Somatic sensory fibres.
(2) Visceral sensory fibres (splanchnic).
(3) Visceral motor fibres (splanchnic).
(4) Somatic motor fibres.

Moreover, this establishes the connexions with the spinal cord of the somato-pleure and splanchnopleure, an essential step before an attempt is made to interpret the phenomena of abdominal pain. The method to be adopted is to study clinical facts and to explain the symptoms and signs present in given cases as far as they may be accounted for by the nervous connexions of the viscus involved by disease. Mackenzie's work opens up the conception that tion that the wide-spread phenomena are the result of a disordered viscus stimulating the cells within the spinal cord in that segment with which the viscus is connected. How this actually occurs and what is the adequate stimulus to produce this effect is not fully explained. Moreover, as late as November, 1917, speaking to the Edinburgh Pathological Club, Mackenzie (4) makes the following statement referring to a case of gastric ulcer:—

> Here we get the effects of a stimulation of the central nervous system; the stimuli are far more delicate and far more in accordance with natural processes than a physiological experiment. We get pain of a very definite kind; altered sensibility of the skin and other structures of the external body wall, contractions of portions of the abdominal muscle of a peculiar kind, which is scarcely yet recognized and reflex effects upon remote organs stimulating them into activity. The regions in which these phenomena occur and the manner in which they spread, point to the fact that there are paths in the spinal cord of which neither the physiologist nor the neurologist have any conception.

I shall endeavour to employ illustrative cases in order to demonstrate which stimulus is adequate to produce the central disturbance and to elucidate the paths present within the cord.

In the first place it is necessary to investigate the patient's history of pain separate from any associated phenomena.

II.—Abdominal Pain: The History of the Patient.

It may be advisable in the first instance to discuss affections of the small intestine, as the innervation of this structure most clearly fits in with the conception already obtained.

It has been shown that the gut wall contains two sets of ganglia with their associated plexuses: Auerbach and Meissner. These are controlled respectively by two sets of fibres:—

(*a*) Sympathetic, from the splanchnic nerves through the superior or inferior mesenteric ganglia. These cause inhibition of the muscular movements.
(*b*) Vagal and pelvic fibres causing contraction.

After what has been said concerning the insensibility of the viscera to ordinary stimuli, the question naturally arises as to the part that these nerves play in the production of pain, which is undoubtedly associated with disturbances of the alimentary canal. A colostomy opening can be cauterized and scratched, that is, it can be acted on by the ordinary stimuli which find response in the case of the extero-ceptive receptors, without sensation. Consequently the adequate stimulus for pain in the case of the alimentary canal must first be determined before the significance of its nervous connexions can be investigated.

Evidence is forthcoming on this point in Mackenzie's case, when he actually saw during an operation without general anæsthetic a vigorous contraction of the exposed intestinal wall. This was repeated several times and on each occasion pain was caused, which the patient declared was located in the region of the umbilicus. Professor A. E. Mills quotes a case in which all the stones were not evacuated from a gall-bladder. Pain was experienced when over-distension of the gall-bladder occurred. A tube introduced into the wound would on such occasion drain away mucus or bile and relieve the pain. Observers have consequently arrived at the conclusion by considering such facts as these, that the normal adequate stimulus for pain in the case of the viscera is associated with increased "internal pressure" or "tension." It will at once be seen that this pressure or tension may become excessive either as a result of over-distension or of over-contraction of the wall of the hollow viscus.

In the case of the alimentary canal this condition could readily be caused by a disturbance of the normal mechanism of peristalsis. This movement is an example of the law of reciprocal innervation. The contraction of one portion of the gut wall is accompanied by inhibition of the muscle in the portion beyond. Such phenomena are controlled by the interaction of two reflex arcs. The first arc is well shown in the accompanying diagram (Figure III.)

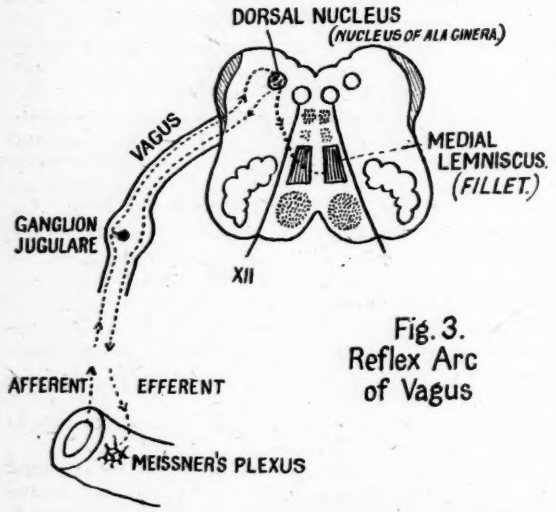

Fig. 3. Reflex Arc of Vagus

from Gaskell (5). This arc is wholly vagal. The sensory cell is situated in one of the ganglia of the vagus. The peripheral fibre comes from the gut wall. The central process passes to the receptive nucleus of the vagus. Here an intermediate cell connects the afferent fibre with the excitor cell, which passes from this nucleus of the vagus to the gut wall. It may be said that the adequate stimulus for reflex action through this arc is a dilated, stretched muscle wall. Within normal limits the bowel can prevent over-distension by the action of this arc and no sensation results. If, however, over-distension should occur, the stimuli will be so intense that a second neurone in the vagus nucleus will become involved. I believe that the axone of this cell passes to the medial lemniscus or fillet of the opposite side and thence to the thalamus. Here or at the cortex a sensation of flatulence results. Collateral evidence supporting this idea is that in bulbar palsy in which the vagus nucleus is involved, the sense of flatulence is lost. Normally, however, before flatulence occurs the reflex arc involved leads to contraction of the muscle wall of the gut. A contracted wall now stimulates the second reflex arc.

The second arc is wholly sympathetic. The afferent fibre passes from the gut wall through sympathetic plexuses until it forms an afferent fibre in one of the splanchnic nerves. The cell body is in the spinal ganglion of a spinal nerve. The peripheral fibres pass to the spinal nerve either by the white or grey ramus and then the central processes pass into the posterior funiculus and thence to the sympathetic group of cells in the posterior horn near its base. Johnston (6) says that this group of cells in fishes corresponds to Clarke's column from which a second relay passes to the cerebellum on the same side by the direct cerebellar tract. Moreover, terminal afferent fibres are seen to end in relation to cells with very short axones which almost immediately

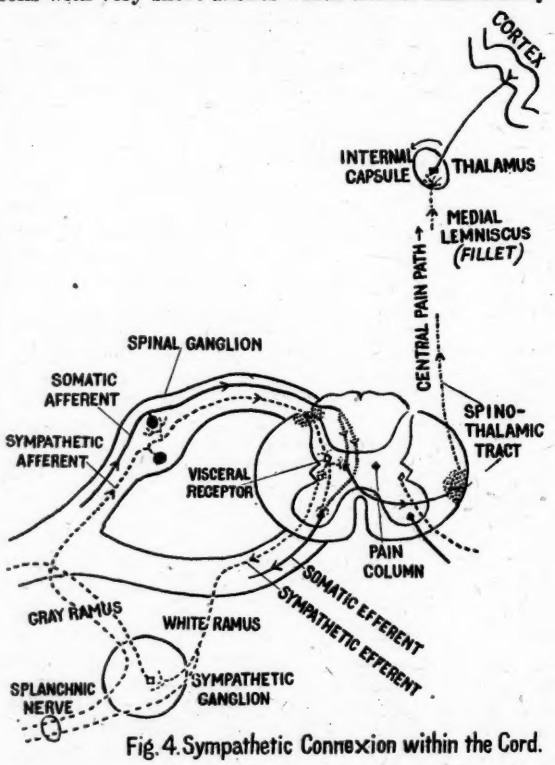

Fig. 4. Sympathetic Connexion within the Cord.

subdivide—Golgi's type II. cells. Other cells of this type have been traced actually beyond the receptor nucleus to the effector nucleus (excitor splanchnic nucleus) situated in the lateral horn in man, and a viscero-motor reflex is thus set up. This is therefore a short segmental reflex for inhibition of the gut wall. The longer reflex through the cerebellum will co-

ordinate the movements of an extensive portion of the gut wall. The short reflex controls a short segments of the gut (Figure IV.).

Edinger describes sympathetic afferent fibres which send their central processes to the somatic sensory cells of spinal ganglia and arborize about the cell body. It appears to me that these are simply collateral branches of the central processes. They are non-medullated. Ranson (7) has now shown that the proportion of non-medullated to medullated fibres in the posterior nerve root is 3:1. The question arises as to whether these non-medullated are not in part splanchnic afferent fibres. Because they are too numerous to be wholly visceral, Ranson has suggested that they serve for protopathic impulses, but this does not exclude the possibility that some are visceral. They would then end in relation to cells of the *substantia gelatinosa*. However, the second relay visceral afferents may be grouped as the "visceral receptors."

The essential point in regard to abdominal pain is that these visceral receptors are connected by a series of intermediate nerve cells (or collaterals of the axone of one cell) with other cell groups of the grey matter. These connexions are suggested by a consideration of the clinical facts illustrated in the chosen cases.

In the reflex arc at present under consideration, an intermediate cell establishes a connexion with the excitor sympathetic cells of the lateral horn of grey matter. The axones of these cells pass out through the white ramus to reach the gut wall. This arc, when brought into play, produces inhibition of the muscle wall of the gut. The normal stimulus to this inhibition is the tension due to the contraction of the gut wall brought about by the vagus. The presence of foreign bodies or inflamed mucous membrane, however, may so stimulate the reflex arc through the vagus that dilatation cannot result. The intensity of the inhibitory stimuli for dilatation now increases and the synapse between the afferent fibre of the sympathetic arc and a new intermediate cell is opened up. This intermediate cell in turn branches around cells of the pain group of the posterior horn of grey matter. Stimulation of these cells gives rise to impulses which are conveyed by crossed spino-thalamic paths to the thalamus and eventually through the internal capsule they reach the sensory cortex. Pain now results at the cortex or thalamus or both. As this group of pain cells is the same as that stimulated by impulses from the skin connected with the spinal nerve concerned, pain is often referred by the brain to this portion of the abdominal wall. This would explain Mackenzie's case. A spinal connexion between the splanchnic and somatic nervous systems is assumed to exist. The following case is also an excellent illustration of how the nervous arcs normally involved in the mechanism of peristalsis can by their connexions give rise to pain, should that mechanism become disordered.

CASE I.—*Intestinal Obstruction.*—The first immediate operation witnessed by the writer was a case of almost complete intestinal obstruction. For three days prior to the operation symptoms of an abdominal crisis were manifested. Intermittent but severe pain had been referred to the navel during this period. The patient had vomited several times (the character of the vomit is not recorded). Occasional periods of relief intervened. Throughout the patient tended towards constipation, but this was not absolute. Early on the third day the symptoms became more severe. The vomiting was marked. Pain in the region of the umbilicus was constant. The patient became pale and anxious looking, the breathing shallow and the pulse soft and rapid.

A careful history revealed that the patient had experienced periodic attacks of pain in the region of the right costal margin for several years. The pain was also felt in the region of the right scapula. No jaundice had occurred. The case was diagnosed by the surgeon as intestinal obstruction, due to gall stones and on operation a mass of green mucoid character containing gall stones was removed from the jejunum.

The history of pain may be explained in terms of the foregoing arguments as follows:—

The first onset of pain in the illness which led to operation, came with the entrance of a foreign body into the lumen of the alimentary canal. The alternating movement of relaxation and contraction were interfered with by this body and the segment of the gut, for the time being containing it, was stimulated to contraction which was maintained by the vagus. Stimuli now passed from the gut wall along the afferent sympathetic nerve fibres to the dorsal root of the 10th thoracic nerve. The impulses passed to the visceral receptor cells which in turn became connected with local association neurones and these neurones transmitted the impulse to the sympathetic efferent cells of the lateral horn. Passing through the white rami to the gut wall the axones of these cells conveyed efferent impulses for relaxation of the gut. This, however, could not occur where the first vagal reflex arc had been over-acting. The intensity of the afferent stimuli was now much greater than normally and on reaching the cord, it caused a new synapse to be opened between the visceral receptor cell and the association neurone connected with the pain column. The group of pain cells was stimulated and impulses passed by way of the spino-thalamic paths to the thalamus and thence to the brain. Here the sensation of pain was referred to the area of skin (the umbilical region); which usually stimulates the group of cord cells; pain was therefore felt "at the navel." As the foreign body was forced on, relief from the pain occurred as the stimulating substance entered a widely dilated portion of the gut. This relief is dependent on the action of the sympathetic cerebellar reflex. Vagal stimuli again caused contraction in the region of the body and the process was repeated. The exacerbation of symptoms was due to a more permanent spasm of the gut wall.

The vomiting in the early stages may be explained on the assumption that the stimuli to the vagus nucleus were so intense that an overflow occurred, which stimulated the cells of the vomiting centre by breaking down the resistance of the new synapses. The stomach and the diaphragm were thus stimulated to contraction.

Moreover, the sympathetic inhibitory reflex would be set in motion proximal to the site of the foreign body by the contraction of the wall caused by vagal stimuli. Dilatation cannot result in the proximity of the body, but at a point remote from the site the less efficient contraction resulting from the vagal reflex could be inhibited. This would give rise to contraction just proximal to this; the contraction in turn would be inhibited. In this way a wave of alternate

dilatation and contraction would be produced extending proximally—anti-peristalsis. This would explain the stercoraceous vomit of these cases.

The pain history prior to the expulsion of the stones from the bile ducts in the preceding case, is further illustrated in the following history:—

Case II.—Two years ago the patient, a man of middle age, had a severe attack of pain accompanied by vomiting. At first the "pain was everywhere," but later settled down "under the breast bone." There was no pain under the right scapula, but the pain seemed to penetrate through to the posterior aspect. It was in front "dead in the mid-line." He received treatment and recovered. A fortnight before operation he again had severe pain, "which seemed to spread in from all sides to settle finally in the mid-line under the lower end of his breast bone." The pain later moved slightly to the right side with severe shooting pains under the right shoulder. The pain continued incessantly for two weeks. Since his admission there has been no alteration in its character.

The operative finding was acute cholecystitis. The nervous connexions will again explain how these symptoms could be produced by such a condition. Owing to probably *B. coli* infection, the inflamed mucous lining of the gall-bladder stimulated the vagal reflex into activity with consequent contraction of the muscle wall. But the wall also contains sympathetic nerve fibres. The contracted muscle stimulates the sympathetic afferent fibres which we believe respond to the stimuli due to increased tonus of involuntary muscle. These stimuli determine the passage of impulses by means of the sympathetic afferents to the spinal cord from the seventh thoracic to the ninth thoracic segment. These impulses now pass to the sympathetic efferent fibres to be transmitted as inhibitory impulses to the gall-bladder wall. If this reflex overcomes the vagus reflex, no pain results. But when the vagal reflex produces extreme hypertonicity of the muscle wall, the stimuli to the sympathetic afferent fibres are extremely intense and sufficient to overcome the synapses of the pain group of cells. Pain impulses proceed by the central pain path to the cortex. Here the sensation of pain results. This pain is referred over the whole area of distribution of the seventh to the ninth thoracic nerves as indicated in the history.

Further Radiation of Pain in Biliary Colic.

Pain in gall stone disease may be felt in the right shoulder and may spread down over the outer aspect of the right arm. This has been explained by the view that the phrenic nerve transmits fibres to the spinal cord in the region of the third, fourth and fifth cervical nerves, which set up an irritable focus in this area.

It is probable that the phrenic nerve contains both the afferent and efferent limbs of a splanchnic reflex arc. As a matter of fact, cells in series with the lateral horn sympathetic group are seen scattered in the cervical region of the cord. These, I believe, have a function identical with and supplementary to the efferent fibres of the thoraco-lumbar outflow; they accompany the phrenic nerve to the diaphragmatic sympathetic plexus and thence to the gall-bladder. The afferent fibres may be regarded as non-medullated splanchnic fibres of the posterior nerve roots of the third, fourth and fifth cervical nerves. Impulses for dilatation pass along these fibres to the cervical cord and reflex connexion neurones transmit the impulses to the efferent fibres already described. When the vagus is over-acting, the intense inhibitory stimuli may break down the pain column synapse to give rise, after transmission by the central pain path to the cortex, to the sensation of pain, which is referred to the distribution of the cervical nerves (supraclavicular branches).

The Relation of Gall-Bladder Affections to Gastric Symptoms.

The pain of gall stone disease is frequently liable to be confused with that of gastric affections. This is partly accounted for by the fact that the sympathetic control of the stomach involves the same segment of the cord as that of the gall bladder. In addition, the vagus connexions of the gall-bladder and stomach are probably closely associated. This is because physiologically the intermittent contractions of the gall-bladder and stomach wall are harmonized. On account of this close association overflow impulses from the afferent vagal fibres of the gall-bladder pass into the efferent vagal fibres to the stomach. This causes contraction of the stomach wall. This may produce an actual hour-glass appearance, when the patient is examined by means of the fluorescent screen and the relaxation of this contraction can be observed after the administration of belladonna. The contraction will stimulate the sympathetic afferents of the stomach which transmit inhibitory impulses through the sympathetic reflex arc to the muscle wall. When this fails to cause dilatation of the contracted segment, the hypertonus so stimulates the afferent fibres that a new synapse to the pain column, is, as before, opened up and impulses stream along the central pain path and lead to consciousness of pain. This reinforces the former stimuli and makes the dissociation of gastric pain secondary to gall stone pain and gastric pain, as the result of a primary condition, a matter of moment.

If food be now introduced into the stomach, a further stimulation is added to the vagal reflex. Further contraction occurs. The increased hypertonus stimulates still further the sympathetic reflex for muscle inhibition. The overflow impulses further increase the intensity of the pain stimuli. This is the explanation of post-prandial pain in gall stone disease.

Review of the Mechanism of Peristalsis.

It may be advisable at this stage to review the conception of the mechanism of peristalsis in the light of the foregoing.

Peristaltic movements of the canal occur if all the nervous connexions, except Auerbach's plexus, are severed. The contents of the canal, however, stimulate the vagal reflex to increased action and further contraction of the wall occurs. Dilatation occurs meanwhile just beyond this point as a result of the inhibitory action of the sympathetic arc. As the contents are pushed into this area, they stimulate the vagus to activity and contraction occurs. This pushes the contents on still further. Meanwhile, the first area of contraction becomes dilated. This is due to the stimuli from the contents having disappeared, so

that the vagus no longer holds sway over the sympathetic arc for inhibition. In point of fact, the contracted muscle stimulates the sympathetic afferents and dilatation results through the impulse passing along the sympathetic efferents.

The principles involved in the explanation of pain in the case of intestinal obstruction and of gall-bladder affections may now be utilized to investigate the history of pain in affections of the muscular structures of the uro-genital passages.

CASE III.—*Sarcoma of the Right Kidney.*—C.O., age 50, a labourer, complained of pains in the right side and right lumbar region together with two attacks of hæmaturia and frequency of micturition. Eighteen years ago the patient was lifting a heavy weight of coal when he suddenly felt pain under his right costal margin. He felt severe pain posteriorly, which spread round to his right groin and down in the right side of his scrotum. He passed blood from the penis. After seven days there was no more blood until seven months ago, when he experienced a similar attack to the first. But in the interval he had had occasionally dull pain in the right lumbar region. Six months prior to the second attack the patient noticed a tumour in the region of the dull pain.

The patient was cachectic, having lost 19 kilograms in the last twelve months. He had noticed himself becoming increasingly more breathless on exertion.

An exploratory operation on the right kidney was carried out by separating the fibres of the right rectus. On investigation it was found that the right kidney was considerably enlarged and could not be removed. The transverse colon was adherent to its anterior surface. The diagnosis was sarcoma.

This case demonstrates how acute the symptoms are when muscular organs are affected.

It is a striking example of a condition in which the kidney could be involved without symptoms. The lifting accident simply attracted his attention to the existence of the sarcoma of the right kidney; the lesion of the kidney was manifested by the passage of blood through the ureter to the bladder.

The nerve supply of the ureter is derived from the renal spermatic and hypogastric plexuses. The afferent and efferent fibres of these nerves are connected with the eleventh and twelfth thoracic and first lumbar spinal segments. The distribution of the anterior rami of these nerves in the anterior abdominal wall has already been noted. It will, however, be remembered that the genito-femoral (genito-crural) nerve is a branch of the first and second lumbar nerves.

No nerves pass to the actual secreting cells of the kidney and inflammatory condition,s therefore, cause no pain. Nerves may be traced in the perivascular fibrous tissue of the finer arteries, but their actual termination has not been accurately determined. It will be remembered, however, that vaso-motor changes, increasing or decreasing the blood pressure, regulate to some extent the amount of urinary secretion.

The passage of the blood clots in this case gave rise to the first onset of pain. Increased ureteral contractions resulted from the presence of these clots, probably because their bulk prevented free transmission of the contents in a distal direction. This means that a condition of hypertonus existed. This condition is precisely the one which is the adequate stimulus for sympathetic afferent fibres. The impulses which these fibres transmit to the cord, normally pass by a reflex path through the lateral horn cells back to the ureter.

The arc may be the normal reflex mechanism for ureteral contraction. But hypertonicity will lead to afferent impulses, which open up synapses with the pain cells and travel by the central pain path to the *cortex cerebri*. Pain results. It is largely referred to the distribution of the spinal nerves of the segments concerned. As the clot passes downwards, the more intense stimuli pass to the various segments in succession. Beginning at the eleventh thoracic segment, it ends at the second lumbar segment. This is why the various areas of skin are involved in succession. The frequency of micturition was caused by the irritation of the blood clot within the ureter and later in the bladder itself.

In another case of kidney tumour the same symptoms occurred in a milder degree.

CASE IV.—*Hypernephroma of the Right Kidney.*—The patient, a female, aged 61, complained of periodic attacks of pain in her right side which travelled towards the anterior aspect to the inguinal region of the right side. Her urine was highly coloured during the periods of these attacks. These symptoms had been in evidence for the past nine months without cessation.

On examination both kidneys could be palpated. The urine was sterile on culture, but contained blood. Under a local anæsthetic a cystoscope was passed and indigo carmine (4 c.cm.) was introduced into the sacro-spinalis muscle mass. The ureteral openings were seen to be functionating normally, the urine being intermittently injected into the bladder. In ten minutes the indigo carmine began to be excreted; ureteral catheters were passed and samples of urine collected. The rate of the drip was normal on the left side. It was considerably slower on the right, about one drop each four seconds. Moreover, it contained blood, as was evidenced by the changed colour of the carmine to a dirty brown. The secretory activity of the right kidney and of the right only was impaired. A 5 c.cm. syringe was now connected with the right ureteral catheter and sterile water slowly injected into it. Pain followed this procedure, but the patient could not identify the pain as the one causing her symptoms. This excluded hydro-nephrosis. The catheter was emptied and colloidal silver injected into it for the purpose of taking a ureterogram.

The X-ray examination showed an irregularity of the pelvis of the ureter. The upper four or five minor calyces were replaced by an irregular projecting mass. The lower two or three were regular in outline. This led to the diagnosis of new growth of the kidney involving the pelvis. Nephrectomy was performed and on histological examination the tumour proved to be a hypernephroma.

In this case the continuous series of periodic attacks of pain was evidently due to the almost continuous but slow exudation of blood from the tumour into the pelvis. No large clots were formed and severe symptoms did not ensue, because of the easy escape for the effusion. The mild irritation of her bladder wall caused almost constant increased frequency of micturition. Nephrectomy removed the symptoms.

The third urinary case is of an entirely different character; the constancy of the pain was different and it revealed no tendency to move down beyond the right iliac fossa.

CASE V.—The patient, a female, aged 25, school teacher, was seen almost immediately on admission into hospital. She complained of pain extending from her right costal margin in the posterior axillary line to the level of the upper margin of the iliac crest in front. She had had scanty micturition for some days.

On examination the patient was found to be tender a little lower than McBurney's point. Inquiry elicited the

information that her appendix had been removed some years before. No rigidity of the muscles could be perceived. The skin in this region was found to be hyperæsthetic on being pinched. This test could be repeated as far as the lower two ribs, that is, mainly along the course of the eleventh thoracic nerve. No hyperæsthesia was noticeable on the crest of the ilium.

On palpation during inspiration the right kidney was found to be tender. No enlargement could be made out. The provisional diagnosis was hydro-nephrosis. An X-ray diagnosis was negative for stone. The surgeon had difficulty in reaching the pelvis with a ureteral catheter. The pain reproduction test was positive. The diagnosis was confirmed at operation.

The symptoms suggested irritation of the pelvic wall without any foreign body passing into the ureter. The increased intensity of the stimuli from the pelvis caused the opening up of the path to the cells of the pain column, which are in association with the pain fibres of the lower thoracic nerves; the impulse transmitted to the brain caused a referred pain in the abdominal wall, along the course of the nerves. When this nerve is stimulated by palpation, two sets of impulses simultaneously reach the cells of the pain column (Figure V.):—

(*a*) From the pelvis of the ureter.
(*b*) From the skin.

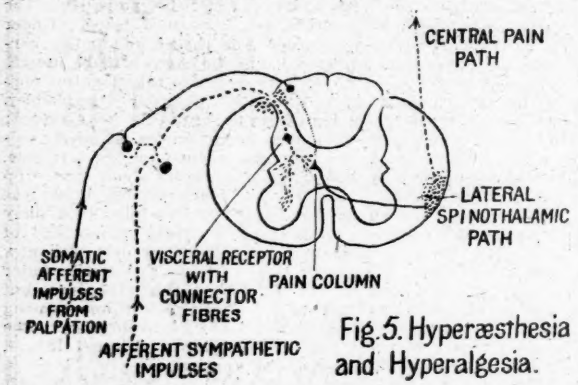

Fig. 5. Hyperæsthesia and Hyperalgesia.

There is then a numerical increase in the afferent stimuli to the second relay neurones. This leads to hyperæsthesia over the area of supply of the nerve concerned and this may be evident even when visceral impulses cannot of themselves produce pain. Numerical increase in the sympathetic nerves involved from an extensive area of muscular contraction, may, moreover, be the factor which ordinarily gives rise to the breaking down of the synapse with the second relay neurones of the central pain path in order to cause visceral pain.

Case VI.—*Salpingitis.*—A female, aged 26, complained of constant dull pain in the left iliac fossa. She had had a "section" several years previously. Nothing abnormal was detected through the vagina. On operation it was found that at the previous abdominal section the left ovary only had been removed. The left tube was still *in situ*. On examination the left tube was seen to be bound down by fine adhesions to the parietal peritoneum. No adhesions were present elsewhere.

The fine adhesions indicate that infection was present in the left tube. It is difficult to understand how fine adhesions *per se* could cause pain. As will be seen later, adhesions probably cause pain by allowing of the stretching of the parietal peritoneum.

The tube, however, was an inflamed tube, with an inflamed mucous membrane. The stimuli from this foreign mucous layer evidently caused reflex contraction through the sympathetic arc and caused hypertonicity. This hypertonicity determined the passage of impulses along afferent sympathetic fibres which broke down the synaptic connexions between the visceral afferent receptors and the pain group of cells. This group is normally stimulated by pain impulses from the sensory nerve branches in the left iliac fossa; the cortex, on receiving pain impulses, may refer them to the distribution of these somatic nerves.

(To be Continued.)

BACILLARY DYSENTERY.[1]

By **James Bentley, M.B., B.S. (Edin.),**
Superintendent of the Hospital for the Insane, Claremont, Western Australia.

When asked to read a paper before this Branch of the British Medical Association I was undecided as to what subject to select. As I have been interested in bacillary dysentery for some years and, as this disease is now notifiable in this State, it occurred to me that it might be of some interest to the members of this Association. Further, a number of our soldiers have returned from Gallipoli, Egypt and Palestine where the disease is of common occurrence. It is now a well appreciated fact that carriers of this disease are not infrequent. The possibility of an outbreak of this disease, not only in this State, but all over Australia, gives it more than a passing interest.

Historical.

Dysentery has been a well-known disease from ancient times and has been in all ages a constant companion of war. It prevailed in England in recurring epidemics until the latter half of the last century. At Leeds in 1849 the inspectors found that there were 1,500 cases of dysentery. Although it has largely disappeared as a general disease in Great Britain, it is still the scourge of the majority of its asylums. For example, in 1912, according to the report of the Commissioners of Lunacy, there was a total of 1,125 cases and 287 deaths, or a mortality of 25%.

Gettings in an exhaustive report has shown that Wakefield Asylum was opened in 1818 and a week after a man named Thomas Umpleby was admitted who was suffering from dysentery. The disease has been practically continuous there ever since, so that the disease as it exists to-day, is probably descended from the progeny of his bacilli.

I will only mention that asylum dysentery is now generally regarded as due to the *B. dysenteriæ*, of which there are many forms and five main types. The details of these different bacilli are not in my province.

About 1911 I suggested to the late Dr. Steel, who was then Bacteriologist to the Public Health Department of Western Australia, that a number of patients in this institution were affected by a disease which on clinical grounds resembled bacillary dysentery. His co-operation was sought in the isolation of the organ-

[1] Read at a Meeting of the Western Australian Branch of the British Medical Association on May 25, 1920.

ism. Dr. Steel willingly undertook this examination and isolated a bacillus of the dysenteric group, but the actual type was not definitely identified, as he relinquished his post. However, as his investigations confirmed our clinical diagnosis that the source of the disease was bacillary and as anti-dysenteric serum is polyvalent, it was then adopted as a routine treatment, with results which you will presently hear.

Infection.

From an infective point of view dysentery is to be regarded as in the same category as typhoid fever. Unhygienic surroundings, although they may be a contributory cause, are not to be regarded seriously in the spread of this disease. The chief channel is infection, either directly or indirectly, from one patient to another. When all the evidence is weighed, the chief means which is likely to cause its spread in this country is the "carrier" or else directly from a patient suffering from dysentery.

Shiga writing on this subject states: "Through the winter there are only a few cases and from some parts of the country it disappears entirely, only to reappear in the next year." We can therefore suppose that the dysentery bacilli exist through the winter in the human subject. How otherwise can the disease be carried on from one year to the other. McAllister has supported this by agglutination tests, but I can find no evidence in the literature to prove this fact conclusively. Momose has stated that the bacilli usually disappear from the stools in 13 to 15 days. According to Shiga and Ohno the lighter the attack, the sooner the bacilli disappear from the stools. These observers have found them in the stools 19 days after convalescence.

Of course, there are other means of spread, such as the pollution of water and milk. It has been shown that an epidemic was caused in Japan by persons bathing in infected water. I often wondered how much of the dysentery which occurred at Gallipoli was due to bathing in water which must have been infected, as most of the troops suffered at one time or another from dysentery. It would also be of interest in this respect to ascertain if the bacillus can flourish in sea water.

It is probable that the fly has a part in the spread of this disease where it has access to the stools. It has been shown by a commission at Salonica that the fly can carry the bacillus on its feet and that its dejecta contains bacilli for at least 24 hours after it was fed on a culture.

In English asylums dysentery is most common from August to January, during which time it is nearly twice as frequent as in the other six months of the year (1914 report of the Commissioners of Lunacy). In the Claremont Hospital for the Insane, the disease is almost equally prevalent all the year round. This distribution is probably accounted for by the more equable climate of this State as compared with that of Britain.

General Symptoms.

Many of the cases begin with general symptoms in this institution. The disease is frequently ushered in by headache and malaise. At this time the temperature is elevated to a moderate degree, usually 38° or 39° C., In only two cases did the temperature reach 40° C. or over on the first day; one of these patients, aged 68, died and the other recovered. There may be slight abdominal pain. If the abdomen is examined at this stage, no rigidity is found, but there is definite tenderness most frequently over the sigmoid and splenic flexures of the colon. This is of the utmost diagnostic importance. Cases are not infrequently diagnosed as dysentery in this institution before there has been even one loose stool; they have subsequently turned out to be typical cases of this disease.

Definite abdominal pain and diarrhœa soon develope and tenesmus becomes a prominent symptom. Blood and mucus appear early in the stools. Of 161 cases no note was made of this in 28 cases; if these are discarded, it leaves 133 cases. In 86 of these blood appeared on the first day in the stools, on the second day in 29, on the third in 11, on the fourth in three, on the fifth in two, on the sixth in none, on the seventh in one and on the eighth in one, so that out of 133 cases blood appeared in the stools on the first and second day in 115 cases. The quantity of blood is usually small, but as the disease progresses a larger quantity of blood is occasionally passed.

The stool in the early stages is characteristic. Clear transparent or white mucus flecked or steaked with bright red blood is almost a certain diagnostic sign of bacillary dysentery, according to Major Graham. The blood and mucus are at first mixed with fæcal matter, but the fæcal matter soon disappears and then the stools are small in quantity and consist of only bloody mucus. Shreds of mucous membrane may be present with blood and mucus or the stools may contain a quantity of pus. As the disease progresses the stools may become dirty brown, the so-called "meat-water stools."

Most commonly the disease first makes its appearance in the sigmoid flexure of the colon and the rectum. If the rectum is attacked, tenesmus is a prominent symptom. Less frequently the splenic and hepatic flexures are primarily affected and sometimes the disease spreads to the small intestine. Shiga states that the small intestine may be starting point of the disease. If the small intestine is involved, the toxic symptoms are more marked and the patient's condition more closely simulates a mild "typhoid state."

Nausea and vomiting are symptoms of infrequent occurrence.

Diagnosis.

The diagnosis of dysentery, as a rule, presents no great difficulty. As before pointed out, a rise of temperature, combined with tenderness over the sigmoid or splenic flexure, is in itself sufficient evidence to arrive at a tentative diagnosis of dysentery, even before diarrhœa has set in. Later, when diarrhœa has begun and blood appears in the stools, a differential diagnosis must be made from hæmorrhoids, carcinoma, polypus and syphilis of the rectum.

The diagnosis of bacillary dysentery can be made from amœbic dysentery in the early stages. Blood appears much earlier in bacillary than in amœbic dysentery. It is bright red in colour, whereas in amœbic dysentery it is darker. The general symptoms are more marked in the early stages of bacillary than in amœbic. There are mild cases of bacillary

dysentery which simulate amœbic dysentery and in these the microscope and agglutination reactions have to be made use of and after all these are the only positive tests. A specimen sent for bacteriological examination, is more likely to give positive results in the first two days.

Treatment.

Various methods of treatment have been advocated from time to time; all have been unsuccessful until the advent of serum. Previous to the war there was a general lack of knowledge of this subject. On Gallipoli early in 1915 the majority of cases of dysentery were proved by Ross to be of bacillary origin. In spite of the splendid work which had been done about ten years previously by Shiga in introducing his serum treatment, a divisional order, which established special units for the treatment of this disease, recommended that cases should be treated by small doses of calomel (say 0.06 grm.) twice weekly.

Immediately a diagnosis of bacillary dysentery is made, the patient should be put to bed and isolated in the same manner as a typhoid fever case is dealt with. The diet should be fluid, such as albumin water, barley water and thin soups. These are better than milk. The food should be given in small quantities and at the body temperature. If hot or cold drinks are given, it sets up peristalsis and increases the diarrhœa. It is a good practice to give small doses of magnesium sulphate either alone or combined with sulphate of soda from the start. Great benefit has been reported from this line of treatment in English asylums. A 2% solution of magnesium or sodium sulphate is supposed to prevent the absorption of toxins from the intestinal canal.

Mellanby has shown that if the body fluids are below normal, toxins are absorbed at a maximum rate from the intestinal canal and the absorbed toxins cannot be made harmless by the tissues of the body. This has a very direct bearing on the treatment of dysentery. Graham, working on this fact, treated his patients with intravenous or subcutaneous injection of saline solution and this combined with serum and other treatment reduced the death rate to 0.3%. Fortunately, we have had no cases since he read his paper, so that I have not had an opportunity of putting this treatment to the test, but it seems to supply a long-felt want. The saline treatment should be begun before the tissues have become dehydrated and should be persisted in until all danger is past. Fluid should also be given systematically by the mouth every two hours with the same object in view.

I have tried various intestinal antiseptics, such as salol, but they do not seem to be of any benefit. Lysol enemata have been in use here for some years. These are given as follows: The bowel is first cleared out in the morning by means of an ordinary soap and water enema and half an hour is then allowed to elapse and the first lysol enema is given, consisting of 300 c.cm. of a 6.85% solution of lysol. Later the amount is increased to 600 c.cm. or more thrice daily. The enema is retained for 15 or 20 minutes if possible. This treatment seems rational and at all events relieves the tenesmus and probably destroys a large number of superficially placed bacteria.

Serum Treatment.

While all other methods of treatment are adjuncts, the only known curative treatment is anti-dysenteric serum. This is both bactericidal and antitoxic in its action. When we started this form of treatment in 1912 we gave 10 c.cm. of serum and the result was not very satisfactory—13 cases, with 7 recoveries and 6 deaths. The dose was gradually increased until when I left in 1914 the initial dose was at least 50 c.cm.. During the war there has been a difficulty in obtaining serum and the whole supply had to be indented in England. With a limited supply, only a limited number of cases could be treated and often only with reduced doses. Our experience is that if it is to be used to the best advantage, it must be administered in large doses. At least 60 c.cm. of the polyvalent serum should be administered as an initial dose, to be followed on the next day, if the case is severe, by an equal quantity. By this time there should be definite signs of improvement; if not, a third dose should be given and it should be continued daily until improvement is manifested. If the patient does not show definite improvement, which will be indicated by a diminished number of stools and the disappearance of blood and mucus, the physician should not hesitate to use heroic doses. I have not seen any harm result from these. In recommending this treatment, the subcutaneous method has been used entirely. I have not seen any anaphylactic symptoms, but when some days have elapsed between doses of the serum, calcium chloride or lactate has always been given as a preventive.

I have tried serum *per os* in one case in 1912, when this method of giving serum was recommended in the treatment of diphtheria. The patient died and the method was not further continued.

The earlier serum is given, the better are the results.

Shiga states that the mortality, after treatment with drugs, is from 22% to 26%. If serum is employed the mortality is reduced to 9% to 12%. With other treatment, but without serum, in this institution the mortality has been 38% of cases. With serum in 1918 and 1919 in this institution there were 28 cases with 2 deaths, or a mortality of approximately 7%.

Serum also shortens the length of the attack in patients who recover.

The main object of these remarks is to draw attention to the early diagnosis of bacillary dysentery. As soon as a clinical diagnosis is made, a dose of polyvalent anti-dysenteric serum should be given. When any one of us diagnoses a case of diphtheria, we immediately give the appropriate serum. Why should we not look on dysentery in the same light?

Reviews.

THE ADMINISTRATION OF ANÆSTHETICS.

Dr. Barton's book, "Backwaters of Lethe,"[1] consists of a series of eight essays in which he sets forth his views on the subject of anæsthetics in a light and readable manner. In the opening chapter the status and emoluments of the anæsthetist are discussed and the particular mental and physical endowments which make for proficiency in this branch of practice are indicated. In the succeeding essays the author describes in detail the particular instruments

[1] Backwaters of Lethe: Some Anæsthetic Notions, by G. A. H. Barton, M.D.; 1920. London: H. K. Lewis & Co., Ltd.; Crown 8vo., pp. 151, with 11 illustrations, including two plates. Price, 5s. net.

and apparatus which he has found most useful in practice. It may be remarked in this connexion that the success of any method of anæsthetization depends quite as much on the personal equation as on the particular variety of apparatus employed. With regard to the administration of chloroform stress is laid upon the danger of manipulations and painful stimulation while the patient is in the early stages of narcosis, before a condition of full surgical anæsthesia has been induced. The writer advocates the use of alkaloids, such as morphine and atropine or scopolamine, as a preliminary to general anæsthesia, but points out very properly the contraindication for morphine in operations in which blood is liable to collect in the pharynx. As a routine method of anæsthesia the "C.E."—ethyl chloride—ether sequence is advocated, proceded by the use of alkaloids, the employment of the author's own particular type of inhaler being described in detail. In the section on nitrous oxide the older forms of apparatus are described, no mention being made of the recent developments of the Coxeter portable outfit with sight feed, or the well-known Ohio apparatus, which is in common use in America, and possesses the advantage of an accurate percentage control of the vapours. In discussing modern methods reference is made to Shipway's apparatus and it is rightly pointed out that the value of warming by a U tube in a thermos flask is negligible unless the vapour is also moistened by being passed over the surface of hot water. The question of the value of warming the vapour is well discussed and the opinion is expressed that in view of the low specific heat as gases and vapours warming is hardly necessary.

The intravenous and rectal methods of ether administration are briefly and tersely described, as is also spinal anæsthesia. Little mention is made of the advances that have been made by the introduction of motor blowers to deliver ether vapours of constant percentages for intrapharyngeal and intratracheal administrations, nor is the field of utility for these methods in the surgery of the chest and upper air ways fully elaborated. The final essay contains many useful hints calculated to guide the inexperienced in dealing with many of the difficulties which may beset the anæsthetist.

APHASIA.

As Dr. Frederick Tilney aptly remarks in the preface to Dr. Osnato's book,[1] speech, whether it be in its purely linguistic phase, or in the complex mechanism of its production and defects, is a subject which eminently invites controversy. The degree in which one has been imbued by the ideas of Broca or Wernicke, Hughlings Jackson or Marie will determine whether allegiance be with the exact localizer, the extreme diagrammatist, the comprehensive synthetist or the lenticular quadrilateralist. Dr. Osnato does not leave the reader long in doubt as to the ideas which have most impressed him; he is clearly a comprehensive synthetist, but apparently not as familiar as he might be with Hughlings Jackson's numerous contributions to this subject.

The book opens with a presentation in abstract of Villiger's work, given to show dissent from Villiger's belief that speech and intellectual development are independent. Attention is then drawn to the teaching of Hughlings Jackson that speech is a part of thought and that in conditions of aphasia thought-processes of all kinds are involved; a teaching repeated by Marie when he stated in his memorable contest with Dejerine that those who deny the intellectual defects shown by aphasics, must have neglected to use even the simplest of intelligence tests. Here we have the keynote of the writer's argument, part of which is to compare aphasia with apraxia, in the endeavour to prove that speech is a form of skilled voluntary movement and a function of the entire intellect; therefore the development of what we know as general intelligence is inalienable from the development of speech and skilled voluntary movement. Moreover, speaking clinically, aphasia and apraxia, as disorders of intelligence, are inseparable and indivisible.

[1] Aphasia and Associated Speech Problems, by Michael Osnato, M.D., Preface by Frederick Tilney, M.D., Ph.D.; 1920. New York: Paul B. Hoeber, Demy 8vo., pp. 191. Price, $2.50.

Functional disturbances and the influence of mimicry on speech are next discussed with the object of linking up the bearing of intelligence on speech; finally dysarthria, in diseases such as Friedreich's ataxia and multiple sclerosis, is pointed to as indicating that the cerebellum participates with the brain in the speech hierarchy.

Many years have passed since the master mind of Hughlings Jackson gave us the dictum that "the loss of speech is the loss of power to propositionize." It is also long since Marie emphasized the loss of intelligence demonstrable in the aphasic. Therefore, in the book before us no new ground is broken. The work is really a stimulating review, from a special standpoint and under dogmatic inflection of hypotheses concerning speech production and its defects. The contained speculations rest mainly on a clinical basis, small pretence being made to knowledge of the anatomy and physiology of the subject. The frequent mutilation of the names of classical writers points either to lack of first-hand acquaintance with their work, or to the holding of a paragraphic pen.

Congress Notes.

The date of the Australasian Medical Congress is August 23-28, 1920.

The official address of the Congress is: The Honorary Secretary, 11th Session, Australasian Medical Congress, B.M.A. Building, Adelaide Street, Brisbane.

The following are the Local Secretaries in the several States:—

***New South Wales*: Dr. F. Brown Craig, Macquarie Street, Sydney.**

***Victoria*: Dr. A. Leo Kenny, Collins Street, Melbourne.**

***South Australia*: Dr. F. S. Hone, North Terrace, Adelaide.**

***Western Australia*: Dr. W. Trethowan, 267 St. George's Terrace, Perth.**

***Tasmania*: Dr. E. Brettingham Moore, Macquarie Street, Hobart.**

***New Zealand*: Dr. G. Bruton Sweet, Remuera Road, Auckland.**

***Queensland*: The Honorary General Secretary and the Coadjutor Secretary, c/o. Queensland Branch, British Medical Association, Adelaide Street, Brisbane.**

The following is a list of the Honorary Secretaries of the Sections:—

(*i.*) *Medicine*: Dr. Andrew Stewart, Wickham Terrace, Brisbane.

(*ii.*) *Surgery*: Dr. Donald A. Cameron, Wickham Terrace, Brisbane.

(*iii.*) *Obstetrics and Gynæcology*: Dr. Lillian V. Cooper, George Street, Brisbane.

(*iv.*) *Pathology and Bacteriology*: (In place of Dr. A. W. Dean) D. J. V. Duhig, Wickham Terrace, Brisbane, and Dr. A. Breinl, Institute of Tropical Medicine, Townsville.

(*v.*) *Public Health*: Dr. J. S. C. Elkington, Federal Quarantine Department, Brisbane.

(*vi.*) *Ophthalmology*: Dr. J. Lockhart Gibson, Wickham Terrace, Brisbane.

(*vii.*) *Otology, Rhinology and Laryngology*: Dr. W. N. Robertson, Wickham Terrace, Brisbane.

(*viii.*) *Diseases of Children*: Dr. A. Jefferis Turner, Wickham Terrace, Brisbane.

(*ix.*) *Naval and Military Medicine and Surgery*: Dr. G. P. Dixon, C.B.E., Wickham Terrace, Brisbane.

(*x.*) *Neurology and Psychological Medicine*: Dr. T. H. R. Mathewson, Brunswick Street, New Farm, Brisbane, and Dr. J. R. Nicoll, Hospital for Insane, Toowoomba.

(*xi.*) *Dermatology and Radiology*: Dr. V. McDowall, Preston House, Queen Street, Brisbane.

The subject of Professor W. A. Osborne's public address, which will be delivered at 8 p.m. on August 25, 1920, in the Exhibition Hall of the Technical College, will be physiological factors in the development of an Australian race.

Section of Public Health and State Medicine.

The Honorary Secretary of the Section of Public Health and State Medicine announces that, in addition to the list of papers published in *The Medical Journal of Australia* of July 17, 1920 (page 68), the following papers will be contributed:

(*ix.*) Industrial Hygiene as Applied to Women Munition Workers.
(*x.*) The Relationship of the General Practitioner to General Medicine.
(*xi.*) The Teaching of Public Health and Preventive Medicine to the Medical Student.
(*xii.*) Legislation and Administration with Reference to Venereal Disease.
(*xiii.*) The Control of Venereal Disease in the Mercantile Marine.
(*xiv.*) The Organization and Operation of Venereal Disease Clinics.
(*xv.*) Venereal Diseases in North Queensland.
(*xvi.*) Developments in Australian Maritime Hygiene Under the *Navigation Act, 1912-1919.*
(*xvii.*) The Application to Civil Life of the Lessons of Military Hygiene Derived from the Great War.
(*xviii.*) Team Work in Sanitation.
(*xix.*) The Control of Insect Vectors in War and Peace.
(*xx.*) Malaria in Palestine: Experiences with a Field Laboratory.
(*xxi.*) Anti-Malarial Work in Sinai and Palestine with the Anzac Mounted Division.
(*xxii.*) The Statistical Survey of the Causes of Death in Australia.
(*xxiii.*) The Health of the School Children in the Far North of South Australia.

The chief topics of discussion in this Section will be preventive medicine in its relation to tropical Australia and the administrative aspects of the endeavour to control venereal disease.

It is hoped that the members taking part in the discussions will devote special attention to the subject of preventive medicine. It is important that there should be free association between those engaged in official administrative duties connected with preventive medicine and the general practitioner, who has the opportunity of applying preventive measures to individuals exposed to risk. Professor Osborne will read a paper on some new aspects of the function of the skin in temperature regulation. This paper will probably be read at the combined meeting of the Sections of Medicine and of Public Health.

Travelling Arrangements.

The following notice is issued for the benefit of members travelling from Victoria to the Congress:—

The first class single railway fare from Melbourne to Brisbane for a member of Congress and his wife, accompanying him, is £5 for each person. To obtain this reduced fare, each person must present at the booking office in Melbourne two vouchers, to be obtained from the Congress Secretary in Victoria, 13 Collins Street, Melbourne, not later than Saturday, August 14, 1920. The member can leave Melbourne on Tuesday (August 17), Wednesday (August 18), Thursday (August 19), Friday (August 20), or Saturday (August 21). The journey may be broken for one night only in Sydney on both the forward and the return journey. No return tickets are issued. The tickets may be purchased and sleeping berths reserved for the through journey to Brisbane at the Tourist Bureau, Collins Street, or at Spencer Street (foot of Bourke Street) fourteen days before the date of the commencement of the journey. To secure the concession for the return journey, the return voucher, previously stamped at the Melbourne booking office, must be presented at Brisbane. The return journey must be begun not later than September 23, 1920. Members residing in country towns in Victoria can obtain from the Congress Secretary for Victoria a voucher entitling the holder to purchase a return ticket from the country town to Melbourne at holiday excursion rates. A separate voucher is required for the member's wife. Country members will further have to secure the vouchers for the journey from Melbourne to Brisbane. These vouchers will be issued not later than August 14, 1920.

Members who live on the North Eastern Line in Victoria must make their applications for the necessary vouchers for tickets from places on this line to Brisbane at an early date. Special notice has to be issued by the railway officials at Spencer Street to the station masters at the stations on this line.

No shipping concessions can be obtained.

The Australian United Steam Navigation Company's *Wodonga* leaves Melbourne on Saturday, August 14, 1920, arrives at Sydney on August 16, 1920, leaves on the following day and arrives at Brisbane on Thursday, August 19, 1920.

The Howard Smith Proprietary, Limited, *Bombala* leaves Melbourne on Wednesday, August 18, 1920, arrives at Sydney on August 20, leaves on the following day and arrives at Brisbane in the morning of August 23, 1920.

The first class single fare from Melbourne to Brisbane is £7 10s.. No return tickets are issued.

We are informed that the dates, fares and ships sailing are subject to variation. Members who wish to travel by sea, are urged to book early.

The latest date for enrolment in Victoria is August 14, 1920.

Organization.

We understand that several practitioners have intimated to the local Secretaries their intention of joining the Congress, but have not yet forwarded a cheque for their subscription. These practitioners are reminded that, until the subscription is paid, they are not members of the Congress and notices concerning the arrangements for travel, vouchers, etc., cannot be sent to them. It is therefore in their interests to send their cheques in as soon as possible.

Obituary.

VICTOR EVELYN COLLINS.

In December, 1913, Victor Evelyn Collins arrived in New South Wales and registered his name as a medical practitioner. He did not remain long in the State. Within six months of his arrival he travelled north to fill a position in the Hospital at Port Douglas between Cairns and Cooktown. Three years later he was appointed Medical Superintendent of the Cairns District Hospital. He retained this position until his death, which took place on July 7, 1920, following an operation for appendicitis.

Victor Evelyn Collins studied medicine at Guy's Hospital in London and qualified with the Diplomas of the Conjoint Colleges in 1896. In 1898 he obtained the degree of M.B. at the London University. Seven years later he was successful in gaining his doctor's degree. In the early part of his career he sought fresh fields in South Africa. Later he travelled to Hawaii and thence to the East. His professional attainments were considerable and he would probably have achieved more, had his restlessness not driven him from place to place. He was very reserved, but to those who penetrated this reserve, he was an admirable companion and a good friend. He was as popular among his patients and colleagues as was possible for a man with a considerable degree of reticence. His interest in scientific and sociological problems was deep and his judgement sound. We are informed that at the time of his death he had nearly completed a contribution intended for the discussion on tropical Australia at the Australasian Medical Congress, Brisbane.

In our issue of July 3, 1920, a notice was published to the effect that a large number of medical practitioners in Melbourne had entertained Dr. W. Cuscaden at dinner, for the purpose of expressing their sympathy with him in connexion with the result of his recent action at law. Owing to a very sad accident to a member of Dr. Cuscaden's family, the dinner was postponed at the last moment, at Dr. Cuscaden's request. The proposed dinner has now been abandoned.

The Medical Journal of Australia.

SATURDAY, AUGUST 7, 1920.

The Second Peace Loan.

The Treasury of the Commonwealth of Australia is in need of money. The world is becoming an expensive place to live in or to die in. Individuals have long since recognized that everything costs much more than it did but a short time ago. The tendency is to endeavour to pass on the increased cost to someone else. Fortunately or unfortunately, when everyone adopts the same tactics, the effect in the long run is nil. Some of the increased burdens of to-day may be traced to the obligations of the community to discharge its liabilities to those who bore arms during the world war. The public has to provide money for this purpose and to enable the Government to meet other expenditure incurred during the war. Under ordinary circumstances, when the Government requires money, the individual has to find it out of income. He pays indirectly to the Government by purchasing goods liable to duty or he assists to fill the coffers of the Treasury by paying more for Governmental services. Few realize the extent of their contribution to consolidated revenue or ear-marked departmental expenditure. Income tax, duties, customs charges, taxes, postages, telephone and telegraph charges, railway fares, all yield revenue and to all the average individual contributes a regular but varying amount. At the present time the Federal Government requires the sum of £25,000,000 in order to carry out its scheme of repatriation. The community is not yet compelled to provide this money through any of the channels mentioned above. For the present it is not proposed to collect the money out of the income of the citizens. An opportunity is first to be offered to everyone in Australia to place some of his capital at the disposal of the Government for a period of ten years at a substantial rate of interest. The Second Peace Loan therefore may be regarded as an expedient to obviate the necessity of increased direct and indirect taxation. Australians do not need to be assured that these gilt edged securities are solid and safe. There is no other investment as safe as this one, bearing a rate of interest as high as 6%. We claim, therefore, that it is in the interests of every medical practitioner to assist the Commonwealth to raise the full amount of this loan.

There are other reasons which should induce medical practitioners to apply for bonds or stock. We have the assurance of the Federal Treasurer that the money raised in the Second Peace Loan will be used exclusively for the payment of war obligations and for repatriation purposes. The medical profession stands to-day in a proud position. Its war record is magnificent. No section of the community contributed so large a proportion of its members; no other profession or calling has to its credit so large a proportion of men decorated for conspicuous bravery and distinguished service. The medical profession stands alone in having asked the authorities to apply conscription to itself. The members of this profession will not permit the Government to fail in carrying out its undertaking to reinstate in civil life those who have suffered economically through war service. The Federal Treasurer can be assured that the medical profession will contribute its fair share to the money required for repatriation purposes.

The bonds or inscribed stock can be obtained on convenient terms. Applications will be received between August 6 and September 6, 1920. The applications must be accompanied by either money equivalent to 10% of the total amount or War Loan or First Peace Loan bonds or stock for conversion. The second instalment falls due on October 4, 1920, and the third on November 1, 1920. On each of these dates 20% of the full amount must be paid. A 25% instalment is due on December 6, 1920, while the balance has to be paid on January 5, 1921. It is important to note that War Loan bonds or stock or the First Peace Loan bonds or stock can be offered as payment for 10% of the total amount, provided that the second and subsequent instalments are paid on or before the dates set out. The conversion of the bonds or stock can be effected at par without charge.

In your own interest and for the honour of your country, buy Peace Loan at 6%!

THE FILLING OF CHAIRS.

Professor A. E. Mills recently raised a pertinent question in proposing the health of Professor J. T. Wilson at a farewell dinner at the University of Sydney Medical Society. He asked why it was that we lose so many of our best men. In attempting to find an explanation for the fact, he expressed the belief that the European centres of learning exercised a magnetic attraction to the earnest worker, the seeker for truth. He was satisfied, however, that there must be some other cause at work and he hazarded the opinion that the excessive burdens imposed on our teachers, the impossibility of freeing sufficient time to carry out important research, impell them to accept the less harassed life of a professor in the old country. The thoughts that arose in the mind of the first Australian Professor of Medicine, himself a graduate of an Australian university, when faced with the pending loss to his University of a man of exceptional ability and learning, are reflected in the mind of every practitioner of medicine in Australia who concerns himself with the welfare of his profession. Why do we permit our best men to leave us? This question is inseparably associated with another: Why do we have to search overseas for men to occupy vacant chairs? In our columns last week we published an advertisement calling for applications from candidates for the chair of physiology in the University of Sydney. The applications are to be sent from Australia or elsewhere, not to the University of Sydney, but to a committee of eminent men in London. We have three universities with medical faculties. These institutions have been in existence for many years and are well equipped. The medical schools are large; according to some medical practitioners, too large for the present needs of the Commonwealth. There must be something fundamentally wrong with our methods if we can neither hold the men we import to teach us nor train others to take their place. Professor Mills spoke truly when he referred to the attraction of the European cities to scientists. The attraction, however, is far greater to a scientist from the other side than to an Australian. It has its origin in the greater facility for intercourse with other workers in the same branch of science and in other spheres of learning. We hear much of the isolation of Australia and of the disadvantages of being twelve thousand miles from the great centres. That it exists cannot be denied, but it is not a complete isolation, not a barred door. It is merely relative, the outcome of the smallness of the community of our scientists, anxious to add to the stock of knowledge. The facilities in the old world are greater than our own, because we are foolish enough to limit our resources. We must therefore seek for the real explanation within our own gates, not in the laboratories and class rooms of the old country.

We have held for thirty-three years in the Medical School of the University of Sydney a brilliant anatomist. During this long period we required him to teach a steadily growing number of students, to build up a modern, excellently equipped department of anatomy and to be personally responsible for every division of his department. His leisure time was restricted to a minimum; how he found opportunity for the researches which have made him famous among anatomists, few can imagine. He has received the minimum amount of assistance. In short, his energies were drained throughout the whole period of his service for a relatively small stipend. Little wonder that this system has failed in both directions. We lose Professor Wilson and we look to the old country to find a successor. During his term of office, he has trained many highly capable men. Where are these men? Why is one of them not chosen to succeed him? Each one devoted a period of his career in assisting his erstwhile teacher. Then came the decision—to wait for something to turn up, something, some position, a gauzy, intangible thing. Or to cast off ambitions of attaining eminence in the world of science; to seek better remuneration in the flood light of medical practice. Some of these competent men have achieved eminence in the practical world of surgery and medicine; some have shaken off the dust of Australia from their feet, because of our short-sighted methods. All have fulfilled the promise of their early days, but not as teachers in our own universities. Because the university authorities failed to create ample careers for embryonic professors of the fundamental sciences, it is not worth while for any brilliant student to follow in the footsteps of his master. Because the time of our professors has

been fully occupied with work that could have been achieved by less experienced men, they at length feel it incumbent on them to seek or at least to accept positions of greater freedom and greater opportunity. Because the economic value of a highly trained and competent scientist is not recognized in Australia, we are compelled to engage the services of altruistic men who fail in the world wisdom of demanding a fair recompense for valuable services. The policy of starving our medical schools is suicidal and wrong. We need a great, scientific profession of medicine, not a trade. The highest positions should be those entrusted to the teachers and guides. Attractive conditions must be attached to these positions, not only for the chief actor, but also for his assistants and underlings. Class teaching and the systematic lectures are doomed. They will surely give way to individual teaching. This involves a small army of assistants, who must be offered reasonable remuneration for the important functions they have to fulfil. Until we awaken and brush the mist from our eyes, we shall fail to create self-contained Australian schools of medicine in our Australian universities.

THE HYDROCHLORIC ACID IN GASTRIC JUICE.

Valuable information can be obtained concerning the functional activity of the gastric secretory glands from an examination of the amount of free hydrochloric acid and of the combined hydrochloric acid in the gastric juice. This information is usually applied in a very restricted manner to the problems of disturbed function of the stomach. It is true that many physicians employ an Ewald test meal as a diagnostic measure, but the results of the examination are rarely utilized as a guide to the treatment or as a means of differentiation between the various forms of gastric disorder. The reason is not far to seek. The usual tests applied to the test meal are wholly inadequate and fail to reveal the measure of the departure from the normal physiological activity of acid secretion. Those who carry out the examination often fail to realize that peptic digestion continues only within certain limits of acidity. The acidity must be measured in terms of hydrogen ion concentration. It has been shown that free hydrochloric acid equivalent to pH 1.65 represents the optimum condition for peptic digestion. The old method of expressing the amount of free and combined hydrochloric acid in terms of cubic centimetres, measured by the quantity of tenth normal sodium hydrate necessary to effect a change of colour in methyl orange, alizarin sulphonate, Töpfer's solution or similar indicators, yields results far removed from the measurement of the hydrogen ion concentration. It is further evident that the effect of the combination of acid with protein to form a compound of nearly neutral reaction on peptic digestion is not recognized by the majority of physicians who treat disorders of the stomach.

Biochemists have aimed at the introduction of a method of measuring the hydrogen ion concentration of the gastric juice, which can be applied without elaborate apparatus. The electrometric method is too complicated for clinical application. It is accurate and all new methods must yield results comparable to those obtained by electrometric estimation before they can be accepted. Several methods of measuring free hydrochloric acid by means of chemical reaction have been worked out. Some of these methods are highly ingenious, but none of them are reliable and the experimental error in all is high. It has therefore become necessary to employ one or other of the colorimetric methods to measure the concentration of free hydrogen ions. The chief difficulty lies in the fact that the common indicators either do not change colour suddenly when the fluid passes into the range of neutrality, or the change does not correspond to the variations of acidity met in the gastric juice. Many indicators give false results in the presence of salts or proteins. In addition, it is found that the colour of some indicators in acid solutions fades rapidly. Dr. Alfred T. Shohl and Dr. John H. King have worked at this subject and have devised methods of determining the hydrogen ion concentration of gastric contents which appear to yield accurate results.[1] The test for free hydrochloric acid is based on the fact that the range of the indicator thymol-sulphonephthalein in an acid medium lies between pH 1.2 and pH 2.8, while in an alkaline medium it has a further range of pH 8 to pH 9.6. This indicator is not affected by protein or other buffer substances and has a very sharp end point. The method is extremely simple. The gastric contents, recovered one hour after the test breakfast of a measured amount of water and bread, is filtered or centrifugalized. Of the supernatant fluid 2 c.cm. are placed in one of six standardized test tubes of a given width. To this fluid is added 0.04 c.cm. of a 0.2% solution of thymol-sulphone-phthalein. In other tubes the same quantity of the indicator is placed in acid fluids between pH 1.4 and pH 3.0. The standard dilutions found to match the colour of the gastric fluid with indicator are selected. The actual matching is carried out in a colorimeter. The light is allowed to pass through pairs of tubes. The standard dilutions are placed in front of tubes with a sample of the gastric contents without indicator, while the tube with the gastric contests and indicator is paired with a test tube containing plain water. The light passing through each pair of test tubes illuminates the colour imparted by the indicator, modified by the colour of the sample. The change in colour with small changes in hydrogen ion concentration is very easily recognized. Tests carried out in this manner yielded results approximately agreeing with the measurements of the hydrogen ion concentration by the electrometric method. In practice this method would give information concerning the degree of excess or deficit of acid in the juice.

[1] *Bulletin of the Johns Hopkins Hospital*, May, 1920.

The practitioner could adjust the acidity to approximate the optimum concentration in cases of functional disturbance of the stomach.

In the next place, Drs. Shohl and King, after consideration of the causes of failure to obtain reliable methods for the estimation of combined hydrochloric acid, either in the presence of free acid or in its absence, have arrived at the conclusion that it is useless to endeavour to make a direct estimation. They therefore have worked out a means for measuring the so-called buffer value of gastric contents. By buffer solutions they mean those to which acid or alkali can be added without causing a marked change in the reaction. In the case of the stomach contents, the buffer substance is usually protein, but certain salts also act in this way. When free hydrochloric acid is present, they add one drop of 0.2% alcoholic solution of thymol-sulphone-phthalein to each cubic centimetre of stomach contents. They then titrate with tenth normal sodium hydrate free from carbonate until the full blue colour of the indicator developes. The buffer value is the figure obtained from the result of this titration after subtraction from it of the value of the free hydrochloric acid. When no free acid is present, it is first necessary to ascertain the acid deficit. This is done by titrating with 0.05 normal hydrochloric acid until the pH is just 3.0. It can also be carried out by adding 1 c.cm. of 0.05 normal hydrochloric acid and one drop of the indicator. The value of the free hydrochloric acid is then determined in the colorimeter. The value obtained is multiplied by the number of cubic centimetres of fluid (gastric contents and acid) and the product subtracted from 50 c.cm., the value of the hydrochloric acid added. These methods yield information concerning the acid deficit. To it must be added the alkaline titration value to obtain the buffer value. From this value the acid value of the stomach contents, the acid production and the amount of alkali necessary to bring the contents to the alkalinity of the blood can be calculated. It must be recognized that the buffer value is not identical with the combined acidity, although the values are almost equal. The investigators have controlled their results with these tests and have apparently demonstrated the reliability of the methods employed. All the methods are easy to carry out, require no elaborate apparatus and are quickly performed. They should find a frequent use in the clinic.

PRIMARY PULMONARY TUBERCULOSIS IN CHILDREN.

The question of the portal of entry and method of infection in pulmonary tuberculosis in children has been a controversial matter for some years. Dr. R. G. Canti[1] has recently reported the results of an investigation in cases of children dying from tuberculous infection, undertaken with the object of repeating observations previously recorded by Ghon, in which the lesions found in the lungs were considered to be the primary sites of infection.

It has been maintained that there is a special form of tuberculosis in children, consisting of a primary lung focus, caused by the inhalation of tubercle bacilli. The lung focus is usually small, varying in size from a pin's head to a cherry and most frequently solitary. It is presumed to have origin in the aggregations of lymphoid tissue found in the neighbourhood of the smaller bronchi. The focus, commencing as a small tubercle, soon increases in size and involves the surrounding structures. Caseation and cavity formation may follow, or the process may be arrested at any stage, as evidenced by the presence of fibrosis or calcification. The focus is usually immediately beneath the pleura and predilects the middle of the anterior surface of the upper lobes. The lower lobes are found to be primarily involved in other cases, as is also the middle lobe of the right lung. Infection of the mediastinal glands occurs secondarily. In this connexion a knowledge of the anatomical details of the distribution of the lymphatics of the lungs is of importance. The lymphatic vessels of the middle and lower portions of the lung discharge their contents into the inferior tracheo-bronchial glands, situated in the bifurcation of the trachea. The vessels from the upper lobes discharge into the superior tracheo-bronchial glands, situated in the angle between trachea and bronchus on either side.

With regard to the relation of the lung focus to the mediastinal glands, it is a matter of common observation that if there is a lung focus, there are almost invariably tuberculous glands in the mediastinum. Conversely, if the glands are found to be affected, a tubercular focus in the lungs is usually present. Furthermore, it is noted that the distribution of the affected glands follows the course which might be expected from a knowledge of the anatomical arrangement of the mediastinal lymphatic system. Owing to the intimate lymphatic anastomosis infection may rapidly spread to involve neighbouring groups. If the distribution of the affected glands be examined, it is usually possible to predict the situation of the lung focus. As the disease progresses, fresh lesions occur, prominent amongst which is tuberculous broncho-pneumonia, which may result from the discharge of material from the focus or from the perforation of a bronchus by an adherent gland. The lesion may spread also by direct extension and along the peribronchial lymphatics, or hæmatogenous infection may result in a condition of miliary tuberculosis.

It has been held by many authors that the disease is primarily an infection of the mediastinal glands, arising from the absorption of ingested bacilli from the alimentary tract.

The anatomical distribution of the glands affected, together with the fact that tuberculous mediastinal glands are rarely found without a corresponding focus in the lung, which can often be demonstrated to be of older origin, are arguments commonly adduced against a primary lymphatic infection. It would, moreover, appear improbable that from a primary focus in the glands a solitary focus would evolve in the periphery of the lung, without infection of the intervening tissue.

The supposition that the affection of the lung could be a metastasis from some remote focus is unlikely, since in this case it would be natural to expect to find multiple pulmonary metastases rather than an isolated lesion.

A further light has been thrown on this question

[1] *Quarterly Journal of Medicine*, October, 1919.

by recent study of the type of bacillus found in various lesions.

Since an air-borne infection is derived from the sputum of phthisical patients affected chiefly by bacilli of human type, it is natural to suppose that if the pulmonary lesion in children had resulted from inhalation, the human type of bacillus would be that usually found. Since ingestion tuberculosis is derived almost entirely from milk infected with the bovine type of organism, it would be reasonable to expect that metastatic lesions in the lungs would exhibit this type of infection. Investigation has proved that in cases in which the lesion appeared to have arisen in the respiratory tract, the human type of bacillus was almost invariably found, whereas in cases of primary lesion of the gastro-intestinal tract infection by the bovine bacillus was usually demonstrable.

The summation of these facts constitutes strong evidence in favour of the common exisence of primary pulmonary tuberculosis in children resulting from an air-borne infection.

PULMONARY MANIFESTATIONS IN MALARIA.

The question whether or not a pure malarial infection is capable of producing the symptoms and physical signs of bronchitis, pulmonary congestion and consolidation has been a subject of discussion for many years. The opinions expressed by various authors who are recognized as authorities in tropical medicine, differ considerably in regard to this matter, some believing that the plasmodium is capable of producing actual pneumonic consolidation, whereas many of the more recent writers regard the pulmonary complications which occur, as being due to a superadded infection, usually by the pneumococcus.

In the group of cases sometimes described as pneumonic pernicious fever, in which dyspnœa, cough, bloody expectoration, dulness, bronchial breathing and fine crepitant râles are recognizable clinical features, it has been suggested that these symptoms may be due to a congestion of the lungs, together with a sero-sanguinolent exudation which might result from malarial vaso-motor paralysis. Dr. A. W. Falconer[1] in reviewing observations of a large number of malarial cases, which occurred in the British Salonika Force in 1916, divides the pulmonary complications into three groups: (1) Bronchitic, which was by far the most frequent; (2) pneumonic and broncho-pneumonic, in which there was evidence of a superadded pneumococcal infection; (3) a group in which more or less extensive signs of consolidation appeared without any evidence of superadded infection. All observers agree that pulmonary affections are very frequently associated with severe forms of malarial infection, a bronchitis with varying degrees of pulmonary congestion being noted in a large percentage of all acute cases. It has frequently been observed that one of the most characteristic features of consolidation of the lung in malarial patients is the remarkable way in which the physical signs may vary from day to day. Adequate treatment by quinine often causes a truly dramatic disappearance of symptoms. In such cases it is justifiable to conclude that the physical signs are a direct result of the malarial infection. Massive collapse of the lung, most frequently affecting the lower lobes, appears to be a not very uncommon occurrence in malaria and is generally overlooked or described as pneumonia.

The concurrence of malaria with definite pneumococcal infection of the lung has been recorded by many observers, the pneumococcus having been recovered from the blood stream in such cases. In these cases the patients present the clinical features of lobar pneumonia with typical viscid rusty sputum and well-marked polymorpho-nuclear leucocytosis. Apart from these definite pneumococcal infections numerous cases occur with recognizable changes in the lungs, in which evidence of superadded infection is wanting. In some instances of malarial infection the initial symptoms may be entirely pulmonary, so that the possibility of the presence of the plasmodium is overlooked.

The early recognition of the causative agent in such cases is of great importance, since if untreated by quinine, there is great liability to secondary infection, with the development of true inflammatory changes in the lung. Hence the necessity for a routine examination of the blood for malarial parasites should be recognized in the presence of all febrile diseases arising in regions in which there exists the possibility of malarial infection.

[1] *Quarterly Journal of Medicine*, October, 1919.

THE YOUNG WOMEN'S CHRISTIAN ASSOCIATION IN SYDNEY.

The New South Wales section of the Young Women's Christian Association has determined to ask the public to provide the sum of £37,000 for the purpose of erecting a large, central hostel in Sydney for young girls. Every careful observer will admit that there is abroad at the present time a spirit of licence, of lack of discipline, of defiance of authority, of abuse of freedom among the young which results in the gravest dangers to girls who earn their living in our cities without the protection and guardianship of their parents. This excellent organization has demonstrated to the world that the girl from the country can be raised above the trough of the prevailing immodesty and immorality by providing her with a home and awakening in her a full sense of her responsibilities, both as a citizen and as a member of a small but healthy-minded community. In the Young Women's Christian Association homes or hostels the stranger girl finds comforts, neatness and happiness. She is unconsciously trained to exercise economy, without sacrificing the legitimate pleasures of youth. She is taught to love domestication, so that in the fullness of time she may be fit to undertake the sacred duties of motherhood and to enjoy the delights of marital life. It is repeating the well known to state that this institution has saved thousands of girls from shame, misery and despair. None know the value of these hostels better than the members of the medical profession. We ask our colleagues to support this admirable cause in a practical manner, to enable the promotors to realize their humanitarian aims.

We regret to learn that Dr. Walter Anstice Harvey died, at the age of 77 years, in Hobart on July 28, 1920. The late Dr. Harvey was a student at St. Bartholomew's Hospital and graduated at the London University in 1865. He came to Tasmania in 1887, where he practised for many years. During the last few years of his life he had been living in retirement. He was a member of the Court of Medical Examiners, a body entrusted with the duties of a medical board from 1898 onwards.

Abstracts from Current Medical Literature.

PATHOLOGY.

(44) Streptolysin.

Paul H. de Kruif and Paul M. Ireland have recently studied the production of hæmolysin in serum broth by β-hæmolytic streptococci (*Journ. Infectious Dis.*, April, 1920). In their work the amount of free hæmolysin present at different intervals of incubation has been determined by the titration of the supernatant fluid after high speed (8,000 revolutions a minute) centrifugation of serum broth cultures against constant quantities of washed red blood cells. Lysin production reaches its maximum for the strains studied at a very early period in the life of the serum broth culture. When the serum broth is seeded with young (8- to 12-hour) cultures, this peak is reached at from 7 to 8 hours. When older cultures are used for seeding, the crest may be deferred to 12 hours. The outpouring of streptolysin occurs during the early part of the growth period of the culture. When this growth period ceases the free lysin begins to decrease rapidly, often disappearing completely in 14 hours. When streptolysin has disappeared completely from the supernatant fluid of high speed centrifugation (free lysin) whole cultures may still be hæmolytically active in low titre. This activity is probably due to lysin operating from the surfaces of organisms, at which place it is destroyed less rapidly than in the free state. The comparative efficiency of sheep, horse, human and rabbit serum broth for hæmolysin production has been studied by the authors, who found that heated sera are in general superior to unheated. Sheep and horse serum are distinctly superior in all concentrations to human and far superior to rabbit serum broth. The data gleaned in these quantitative studies have been used in the synthesis of a new blood-agar plate. This plate has been devised with the idea of using the optimum serum concentration for maximal lysin production of a blood which shall be efficient and at the same time accessible to general use. Sheep blood is believed to fill this need. To obtain maximal lysin production high concentrations of serum are necessary (20%). The number of red blood cells is kept down by making artificial combinations of heated serum and cells. The serum-cell mixture is made by combining four parts of heated serum with one part of cells. This mixture is then combined with liquid agar in proportion of one part of the mixture to three of agar. The final concentration, therefore, is: serum, 20%; cells, 5%; and agar, 75%. The proposed medium is not only a very suitable one for demonstration of the hæmolytic zones of β-streptococcus, but is in addition very efficient in the demonstration of multiple concentric zones of green production and hæmolysis for α-streptococcus and pneumococcus.

(45) Lethargic Encephalitis.

In the *Journal of the American Medical Association* (March 27, 1920), Simon Flexner gives an account of lethargic encephalitis. He states that the first cases of the disease recognized in the United States occurred in the winter of 1918-1919 and that there is no reason to suppose that this epidemic affection of the central nervous system occurred previously in America. It is possible to trace the cases of epidemic encephalitis now arising in America to an outbreak in Vienna in the winter of 1916. Cases of the disease occurred in England and France in the early months of 1918 and in America about one year later. The first fatal cases in Vienna supplied, on histological study, a physical basis for the symptoms observed during life; the microscopical appearances in the first English and French cases revealed identical lesions. The anatomical study of cases arising in the United States showed close agreement with the others and now a histological basis of the pathology of the disease, of remarkable concordance, has been provided. On this basis lethargic encephalitis may be regarded as representing a definite pathological as well as clinical complex and as constituting a distinct disease. The histological changes or lesions of lethargic encephalitis may be both extensive and profound. Those so far described as confined to the central nervous system affect particularly the brain and especially the grey matter at the base. While any part of the grey matter may be involved and lesions are found in the cortex and in the cerebellum, the structures particularly affected are those about the third ventricle, the aqueduct of Silvius, the lateral ventricle and optic thalamus and the pons and medulla. The spinal cord is at times involved. In general, the severity of the cerebral lesions diminishes from before backward. The lesions themselves consist of cellular aggregations around the blood vessels, cellular infiltrations in the nerve tissues themselves small, often microscopic, hæmorrhages and an outpouring of plasma or lymph into the tissue interstices. The cellular accumulations and invasions are chiefly mononuclear in nature (lymphocytes, plasma cells, polyblasts); polymorphonuclear cells are also encountered, but are relatively inconspicuous. The lesions themselves occur in nodular and diffuse forms and those of the tissues are at times clearly associated with the vascular affections and at other times are so extensive as not to be brought into relation with particular vascular involvements. The paralyses of the ocular, facial and other muscles which sometimes occur, arise, with rare exceptions, from the cellular and other invasions of the nuclei of the corresponding nerves. The cerebrospinal fluid tends to be under somewhat increased pressure, but clear. The number of cells is slightly increased (very rarely 100 per cubic millimetre) and the globulin content little and sometimes not at all excessive. The cells, which range usually around 10 to 20 per cubic millimetre, consist partly of mononuclear and partly of polymorphonuclear leucocytes.

(46) Anthrax Bacilli Grown on Beef Serum.

J. A. Kolmer, D. C. Wanner and M. E. Koehler publish the results of investigations into the influence of normal beef serum on anthrax bacilli (*Journ. Infectious Dis.*, February, 1920). The authors' interest in the subject was aroused by numerous reports of other workers on the favourable results in the treatment of the pustule and bacteræmia of human anthrax with ordinary beef serum. Inasmuch as rabbits were found highly immune and white mice highly susceptible to the culture of anthrax bacilli employed in this investigation, the serum of these animals was included in the study for the purposes of comparison with beef serum and for the study of the mechanism of the nature of natural immunity to anthrax. Protection tests, consisting in the intraperitoneal injection into mice of heated and unheated normal beef serum in doses ranging from 1 to 10 c.cm. per 100 grm., generally failed to protect mice against 1 to 5 minimal lethal doses of culture. Unheated normal beef serum is bactericidal for the anthrax bacillus *in vitro;* heated serum is somewhat less bactericidal. Rabbit blood is highly bactericidal for anthrax bacilli *in vitro*, while mouse blood is without appreciable effect. The natural immunity of rabbits to anthrax is probably due in part to the bactericidal activity of their blood. Normal beef serum contains variable amounts of agglutinin for anthrax bacilli, the titres ranging from 1:16 to 1:224, rabbit and mouse serum also contains small amounts of agglutinin, the former somewhat more than the latter. Heated serum of the three animals contains somewhat less agglutinin. Normal beef, rabbit and mouse sera do not contain complement fixing substances for antigens of anthrax bacilli. While normal beef serum contains some anti-anthrax substances, they were found without demonstrable protective and curative value in experimental anthrax infections of mice and rabbits.

(47) Diphtheria Mortality.

Dubourg and F. Guénard (*Journal de Médecine de Bordeaux*, February 25, 1920) state that in 1888 the percentage mortality of diphtheria was 54%. In 1890 it fell to 39%, on account of the division of the patients into special pavilions; the erection of separated boxes brought down the mortality to 19.5% in 1894. This is one of the lowest percentages obtained before the discovery of antitoxin. Under the influence of antitoxin, between 1895 and 1917, the average number of deaths has been 7.28%, with a maximum of 15.5% in 1899-1900 and a minimum of 1.53% in 1907. The number of interventions for croup has also diminished considerably; in 1894 it was necessary to perform tracheotomy on 56% of children admitted to the hospital, whereas the number fell to 15.34% after the use of antitoxic serum. The authors lay stress on the necessity for large doses and early administration.

PÆDIATRICS.

(48) Water Retention in Pneumonia.

Children who keep up their weight or who gain weight during a febrile disease may begin to lose weight with the disappearance of the fever. In some cases this happens abruptly, in others gradually. This loss is most likely to occur in pneumonia. H. O. Lussky and Hugo Friedstein (*Amer. Jorn. of Dis. of Children*, May, 1920) were able to collect 52 cases of pneumonia suitable for the investigation of this point. In all but one of the 28 cases ending in a crisis there was some loss of weight coincident with the crisis. This loss was frequently quite marked. The average loss of weight of the uncomplicated cases occurring within 24 hours was 272 grammes, with a maximum of 567 grammes and a minimum of 170 grammes in two days, excepting one case, in which no loss was registered. They were unable to find any relationship between the degree and rapidity of the loss to either the height of the temperature, as registered in hospital, or to the duration of the fever. Nor was there a relationship between the range of the drop in temperature and the loss of weight. There were no data to compare the amount of exudate and the loss of weight. The most frequent complication present was *otitis media*. A comparison of the cases of pneumonia complicated by *otitis media* with simple cases of *otitis media* showed that in the former there was a downward gradient in the weight curve from the beginning of the observation with or without a steeper gradient during defervescence. In the latter an abrupt loss of weight with defervescence was the exception rather than the rule. Several factors may enter into this loss of weight in pneumonia, *viz.*, (*i.*) the rapid resolution of a large amount of inflammatory exudate; (*ii.*) the elimination of previously retained water. It has been shown elsewhere that, both in pneumonia and other fevers, there is a marked change in the water economy of the body. There is a general retention during the fever and with remission or discontinuance of the fever an increase in the output of water by all the water excreting organs above the amount ingested. It is possible that in fever the water which is usually at the disposal of the organism for purposes of heat regulation, is no longer available for this purpose. This may occur as the result of the removal of a certain amount of water from the organism or of its chemical absorption within the organism in such a way that it cannot exercise its heat dissipating function by evaporation from the body surfaces and lungs. In such instances it would be possible to have a water retention and still not enough for heat regulation. This may occur in a disease like pneumonia. With restoration of the water-binding power of the tissues to the normal, which may occur more or less abruptly, water would be liberated for heat regulation purposes and elimination in urine. In this way the loss of weight coincident with or shortly preceding the abrupt fall of temperature could be explained.

(49) Abscess of the Lungs in Infants and Children.

Of the fifteen cases of abscess of the lung in young individuals reported by H. Wessler and H. Schwarz (*Amer. Journ. of Dis. of Children*, February, 1920) three followed aspiration of a foreign body, five were subsequent to operation for tonsillectomy, seven could be attributed to pneumonia or other inflammatory lung conditions. The causal organism in all the aspiration cases is probably anaerobic and the determining symptoms appear almost invariably on the thirteenth or fourteenth day after operation or aspiration. The so-called post-pneumonic cases may be separated into two groups: (*a*) the patient apparently contracts a pneumonia which runs an acute course, but on the thirteenth to fourteenth day the classical symptoms of lung abscess appear; (*b*) a broncho-pneumonia, instead of resolving, persists and after a variable time gives rise to a chronic indurative pneumonia, with multiple bronchiestases, which may or may not become gangrenous. Occasionally the condition arises from an empyema. The location of the abscess varies with the ætiology. In the post-operative cases and in the aspiration type of the post-pneumonic abscess, the disease is usually situated in the upper lobes. Abscesses resulting from aspiration of foreign bodies and the chronic broncho-pneumonic type of bronchiectasis are usually localized in the lower lobes. The symptoms consist of a distressing cough, rise of temperature, constant at first, irregular or even absent later. The signs are slight or absent in the early stages; later they become definite, with evidence of cavity formation. On the thirteenth or fourteenth day, signs of gangrene, including fœtid breath, putrid sputum and hæmoptysis are noted. Clubbed fingers appear early and disappear with healing of the abscess. Complications, such as empyema, pyopneumothorax, severe hæmoptysis or cerebral abscess, may appear. The prognosis is bad in post-pneumonic cases, the symptoms usually being manifested for a long time. In post-operative cases, spontaneous cure may occur in about one-third of the cases and then usually within two months. The others usually go from bad to worse, with perhaps frequent remissions in the symptoms. Treatment should be palliative in the post-operative cases for two months, just as with the post-pneumonic type. After this the question of operation arises. Incision and drainage offer less hope for a permanent cure than the very serious operation of lobectomy. The latter operation should be undertaken especially in young individuals. In every case X-ray examination and bronchoscopy should be performed for the detection of a foreign body.

(50) Pneumonia Without Physical Signs.

The question whether a real pneumonia can exist in the chest without physical signs has long been discussed. The best opinion is that in children a positive diagnosis of pneumonia can be made without the presence of physical signs in the chest when there exist several of the following symptoms: fever, over-activity of the *alæ nasi*, a pneumonic type of respiration, with a pause at the end of inspiration, accompanied by an expiratory grunt, a relationship between pulse and respiration approximating 3 to 1, and particularly if there is the added sign of rigidity of the upper extremities, an effort on the part of the child to protect a sore chest. This is of especial importance where an *otitis media* with persistent fever seems to point to a complicating mastoid suppuration, the local symptoms, however, not pointing to mastoid trouble. R. G. Freeman (*Arch. of Pediatrics*, January, 1920) records a series of three cases; signs of consolidation in the chest were entirely absent. Symptoms, however, were very definite and an X-ray examination gave in each case a definite shadow, which cleared as the patient improved. In each case the same area of lung was involved, *viz.*, an area apparently beneath the right axilla. This corresponds to the right middle lobe and in that case signs should have been heard over the anterior portion of the chest. The author considers, however, that the area involved lay in the right upper lobe and that the signs were obscured by the intervention of the right scapula. The paper is accompanied by several excellent X-ray photographs.

(51) Acetonuria in Children.

Of 734 consecutive patients admitted to a children's ward, J. Garland (*Arch. of Pediatrics*, August, 1919) found that the urine of 127, or 17.3%, contained acetone at one time or another, but generally during the first one to six days. The temperature in this group of 127 patients varied from 37° C. to 43.3° C.; every child had some fever. Of this number only two were discharged with the unqualified and sole diagnosis of acidosis. In three or four more a questionable diagnosis of acidosis as a complication was made. The author states that acetonuria does not represent the true clinical disease of acidosis. In the majority of the cases acetone was present for only one day, that of admission; the longest time during which is remained constant in the urine was six days in a case of pneumonia, in which the temperature varied between 39.7° and 41.4° C. for fourteen days. One patient had a true post-operative acidosis. The occurrence of the acetonuria was found to be much more frequent in respiratory than in other diseases. Of the 127 patients with acetone in the urine, only two had true acidosis. The author found that acetone was more frequently present in the urine during pyrexia than when the temperature was not raised. Since acetone is formed from fat, he holds that it is wise to give a diet rich in carbo-hydrates and poor in fats, especially in the early stages of respiratory affections, in order to diminish the production of acetone. Large amounts of liquid are especially indicated, to wash waste products out of the body.

British Medical Association News.

SCIENTIFIC.

A meeting of the Western Australian Branch was held at the Hospital for the Insane, Claremont, on May 23, 1920, Dr. A. T. White, C.M.G., V.D., the President, in the chair.

The President announced that Dr. C. H. Shearman had been granted three months' leave of absence from his duties as Honorary Secretary on account of illness. Dr. T. L. Anderson had been appointed Honorary Secretary.

Dr. A. E. Randell exhibited a gall stone, the shape of the bladder, which had been passed naturally by a woman, aged 72. The patient had been subject to biliary colic.

Dr. W. Trethowan mentioned a case in which a gall stone had been passed into the bowel and caused acute intestinal obstruction.

Dr. J. Bentley reported a case of poisoning with eucalyptus oil. The patient took twelve drops and collapsed. He recovered rapidly.

Dr. W. P. Seed referred to another case of eucalyptus oil poisoning of recent occurrence. The patient was supposed to have taken 30 c.cm.. An emetic had been given and rapid recovery followed.

Dr. R. C. E. Atkinson said that a fatal case had been reported in Western Australia about four years previously. The dose taken was a teaspoonful, *i.e.*, 3.5 c.cm..

Dr. S. J. Cantor said that he had known collapse to follow the swallowing of half a teaspoonful of the oil.

Dr. R. C. E. Atkinson reported the death of two persons after intravenous injection of 0.14 grm. of tartar emetic. Death took place three hours after the injection. He contended that not more than 0.12 grm. should be injected for bilharziosis.

Dr. J. J. Holland said that he has given to a patient 0.06 grm. of tartar emetic intravenously. The patient had vomited. He then gave 0.03 grm.; no vomiting followed. He then tried 0.06 grm. again, but vomiting followed.

Dr. Theodore Atkinson suggested that idiosyncrasy was possibly a factor in fatal cases.

Dr. F. Gill read the notes of a case of fracture-dislocation of a certical vertebra, with paralysis below the level of the lesion. Strong traction was applied and a click was heard. Within twelve hours the paralysis had disappeared.

Dr. J. Bentley read a paper on bacillary dysentery (see page 122).

Dr. A. T. White thanked Dr. Bentley for his admirable paper. He gave a brief account of his own experience of dysentery followed by jaundice.

Dr. W. J. Beveridge referred to the practice in South America of giving pomegranate with cinnamon powder four times a day in dysentery.

Dr. F. A. Hadley recalled five cases of liver abscess following dysentery during the South African war. He also spoke of a case of perforation of a dysenteric ulcer.

Dr. W. Trethowan described the serum treatment of dysentery as carried out at Lemnos. The patients did not do well when the treatment was started after the third day, even if large doses of serum were given. He recommended appendicostomy and lavage of the large bowel in chronic cases.

Dr. R. C. E. Atkinson dealt with the several groups of *Bacillus dysenteriæ*, including the Shiga, the Flexner and the "Y" group and Morgan's bacilli.

Dr. Theo. Anderson spoke of the high mortality prior to the introduction of serum.

Dr. T. L. Anderson described from his own experience the clinical manifestations of infections with *Amœba histolytica* and Shiga's bacillus.

Dr. J. J. Holland asked Dr. Bentley if there was any objection to giving 150 c.cm. of serum as the first dose.

Dr. W. K. Dale gave some interesting particulars of outbreaks of dysentery among the men of the Second and Fourth Armies in France. He stated that *Amœba histolytica* had been found in 5% of soldiers who had not left England.

Dr. G. W. Barber, C.B., C.M.G., D.S.O., Dr. R. E. J. Burns, Dr. W. T. Dermer and Dr. D. M. McWhae, C.M.G., C.B.E., also spoke.

In the course of a short reply, Dr. Bentley stated that abscess of the liver was not a complication of bacillary dysentery.

MEDICO-POLITICAL.

At a meeting of the Western Australian Branch, held on May 23, 1920, a letter was read from the Honorary Secretary of the Federal Committee relative to the payment of personal expenses and travelling expenses of members attending meetings of the Committee.

Dr. W. Trethowan moved that the representative of the Western Australian Branch on the Federal Committee be instructed to support the proposal of the Committee to amend Clause 5 of the constitution to enable it to pay the travelling and personal expenses of its members. The motion was seconded by Dr. J. J. Holland and carried unanimously.

Dr. Theodore Anderson expressed the opinion that no delegate would be able to attend the meetings of the Federal Committee for twelve guineas. He did not know who would undertake to provide the necessary balance. The President pointed out that the Federal Committee proposed to pay both the personal and the travelling expenses of the members attending the meetings.

Dr. J. J. Holland gave notice of a motion as follows:

> That the Federal Committee be asked so to alter their constitution that the Western Australian Branch may vary their representatives at the Federal meetings, owing to the length of time involved in attending these meetings.

Robert Elbury Jefferis, Esq., M.B., Ch.M., 1915 (Univ. Sydney), of Sydney Hospital, has been nominated for election as a member of the New South Wales Branch.

Medical Societies.

MELBOURNE HOSPITAL CLINICAL SOCIETY.

A meeting of the Melbourne Hospital Clinical Society was held at the Melbourne Hospital on June 25, 1920, Mr. Basil Kilvington presiding.

Mr. W. Kent Hughes showed a small boy for whom he was restoring an ear by plastic surgery. The boy's right ear had been torn off during birth by forceps. A plastic cast was shown to demonstrate the condition before operation. The first stage of the operation had been completed; Mr. Kent Hughes outlined the measures he had taken to fashion the new auricle and the means by which he proposed to complete the operation by restoring the lobe.

Mr. Kent Hughes presented a man with an extraordinary tumour of the nose, imparting an expression which he referred to as "frog-face." The man, now aged 61, had been an in-patient of the Austin Hospital for three years, but the condition with which he was affected dated back 22 years. It originated in a blocking of the nostrils, which at first partial, later became complete. After a few years granulomatous growths appeared within each nostril and these now extruded a considerable distance on each side. The patient suffered no constitutional symptoms. His serum did not yield a Wassermann reaction. Histological examination of a portion of the growth disclosed the structure of a myxomatous polypus. Mr. Kent Hughes drew attention to the great expansion of the *alæ nasi* and of the nasal bones. He considered that there would be no great difficulty in giving the patient an air-way, but deprecated any radical and subsequent plastic procedures in view of the man's age. To effect a cure it would be necessary to deal drastically with the antra and frontal sinuses, in addition to the nasal fossæ. He had seen only three examples of "frog-face."

Mr. Leonard Mitchell suggested that an antero-posterior stereoscopic skiagram might indicate the extent of the bony involvement and determine if the air sinus cells were obliterated by polypoid growths.

Mr. Kent Hughes remarked that it was extremely probable that all the mucosa of the nose and accessory sinuses, including that of the ethmoidal and sphenoidal air cells, was a mass of polypi. It was a curious fact, but none the less his experience, that patients with complete nasal obstruction were not so prone to bronchitis and respiratory troubles as were people with partial obstruction.

Mr. Kent Hughes next presented a man with rodent ulcer of 10 years' duration. There was destruction of the whole of the right side of the nose and deep erosion of an extensive area of the maxilla. Treatment by diathermy had brought this very advanced condition to a stage which seemed favourable for skin grafting. Mr. Kent Hughes had elected to employ the Gillies tube method of skin grafting. He exhibited the patient to show the first stage of this procedure, which he had completed.

Mr. Kent Hughes exhibited a second example of rodent ulcer; in this case efforts at skin grafting had been unsuccessful, a fact which he attributed to the deleterious effects of prolonged exposures to X-rays and radium of the skin areas from which flaps were obtained.

Mr. Victor Hurley, C.M.G., presented a man, aged 52, who had first appeared in his out-patient clinic on April 9, 1920. On that occasion he was seen to have sustained a cut in the arm, about mid-way between the elbow and the shoulder. The wound had been sutured. The patient was very much the worse for alcohol. Four days later he reappeared, exhibiting a large hæmatoma which extended from the middle of the arm to the wrist. On examination, the brachial pulse and the radial pulse were absent; there was some anæsthesia over the fingers, but it could not be correlated with any particular nerve distribution. The patient, still under the influence of alcohol, refused to enter the hospital as an in-patient, but came back as an out-patient six weeks later, on account of a burn on the ulnar side of the little finger. The swelling of his arm was then seen to be much reduced; there was definite loss of function in the muscle and some wrist-drop. Anæsthesia of the "glove" type was present up to the level of the wrist; the radial pulse was still absent. With regard to the electrical reactions of the muscles, Dr. Murray had reported that the muscles of the forearm responded to faradism, but reacted only to large currents, on account of the very great skin reaction. No response could be obtained in the intrinsic muscles of the hand, but there was no reaction of degeneration. Mr. Hurley stated that he regarded this patient as having sustained a punctured wound of the brachial artery, upon which had supervened a condition of ischæmic paralysis, similar to that observed after tight splinting. In some remarks relative to this condition Mr. Hurley reminded members that ligature of main vessels was followed, in a certain proportion of instances, by loss of function in the peripherial portion of the limb. All grades were to be observed, from the extreme of gangrene, through ischæmic paralysis, to the establishment of a normal collateral circulation and the return of perfect function. He had notes of five cases in which peripheral defective function had followed ligature of the popliteal artery after gun-shot wounds of this vessel. The importance of the condition lay in its simulation of nerve injuries, by reason of the loss of muscle function and anæsthesia. It had been an interesting observation to note that in many cases of amputation for dry gangrene following injury to the main artery of a limb, the muscles proximal to the line of demarcation were of a pallid colour and resembled those of a rabbit more than those of human beings.

Mr. Alan Newton considered that the points raised by Mr. Hurley indicated the necessity for endeavouring to repair a damaged artery wherever possible, an observation with which Mr. Hurley agreed.

Mr. Basil Kilvington suggested that where there was much extravasation of blood into the tissues, early and free incisions by removing obstacles to the establishment of a collateral circulation, might assist to obviate the conditions to which Mr. Hurley had drawn attention.

Mr. Hurley then asked attention for a man, who two years previously had fallen from a bicycle and sustained an injury to his neck. Paraplegia, affecting the arms and legs, was complete for two weeks, but very gradually improvement commenced and progressed. Altogether the patient spent 10 months in a country hospital and six months after the accident was able to walk moderately well. He had progressed well until a few weeks ago, when he sought treatment at the out-patient department for pains in the arms and shoulders. Mr. Hurley directed attention to the cervical kyphosis, a rounded, not angular curvature and to the contraction in both hands, involving flexion of the fingers into the palm at both interphalangeal joints. All the tendon reflexes were equal and active; there was no sensory disturbance. Dr. Murray had reported that the electrical responses of all the muscles of the arm and shoulder girdle were normal and that there was no evidence of any lesion of the lower motor neurone. In the skiagram exhibited the second, third and fourth cervical vertebræ were seen to be completely and the fifth and sixth partially necrosed. There was no evidence of a tubercular infection. The Wassermann test yielded a negative response.

Mr. Hurley said that he regarded this case as an instance of traumatic rarefying osteitis (Kümmel's disease). This condition was described as following an injury, after which a degree of recovery occurred with the later supervention of pain in the peripheral distribution of the nerves of the affected segments.

Dr. Paul Dane brought forward a man, aged 29, who came under treatment for indigestion of four years' standing. The dyspepsia consisted chiefly of a sensation of fullness after meals and constipation. There had been no pain and no vomiting. The free hydrochloric acid in the gastric content was deficient. There was no occult blood in the stools. On X-ray examination after bismuth meal a hypertonic stomach with delay in emptying and ptosis of the colon was recognized. The arresting feature about this man was his very marked, almost extreme, loss of weight. Influenced by the description of a condition called suprarenal dyspepsia which he had met in the course of reading, Dr. Dane had treated this patient on the basis of suprarenal deficiency. The description given of suprarenal dyspepsia might have been based on the case he had shown. American writers had claimed that some amyotrophies were dependent upon a low adrenalin content in the body. The patient exhibited had been receiving adrenalin for three weeks by mouth and hypodermically. And, although he expressed himself as feeling much improved, Dr. Dane was not yet prepared to draw any conclusion. The man's systolic blood-pressure was 120 mm. and the diastolic 80 mm.. He had exhibited no glycosuria.

Dr. Dane further described the successful treatment of a case of insomnia of twenty years' standing by hypnotic suggestion.

Dr. R. H. Strong introduced a man, aged 69 who was under investigation as to the nature of a tumour in the abdomen. The history given was that fourteen weeks previously the patient had become aware of a hardness in the left iliac fossa. A fortnight later it shifted across to the lower part of the abdomen on the right side and later on appeared to be placed directly under the navel. The patient stated that he had become conscious of a definite lump, which gradually increased in size, but his own impression was that it was slowly diminishing. It had become painful about a month ago and he was obliged to stop work. Dr. Strong demonstrated the abdominal tumour, which was apparent to inspection and extended from the umbilicus downward. The swelling appeared to be superficial. The abdomen moved well with respiration. The tumour was dull on percussion; the fingers could not be made to meet under it and dimpling of the umbilicus occurred when the mass was moved upwards. Extending downwards from the lower pole of the mass to the *symphysis pubis* was a hard, cord-like band, which appeared to be connected with the lower pole of the swelling. The skiagraphist's report was that the transverse colon was diminished in lumen at the level of the mass, moved with it and appeared to be adherent to it. There had been no bowel trouble, no loss of weight and no history suggestive of enclosed urachus.

Dr. J. F. Wilkinson introduced for discussion a man, aged 56, for whom Mr. Syme had performed posterior gastro-enterostomy 14 years before. Prior to this operation, the patient had suffered for several years from flatulence, vomiting and pain after meals. He was subsequently shown to have had a pyloric ulcer, with definite narrowing. Vomiting had supervened four or five hours after meals and often lasted for an hour, but invariably relieved the pain. The relief of symptoms consequent on the operation lasted for 13 years. During the last twelve months pain and vomiting had recurred. The pain was not comparable in severity with the earlier pain. The vomiting, which was occasional, relieved it. The total acidity, as determined by a test-meal examination, was 36 and the free hydrochloric

acid 17. The radiographer's report on the X-ray examination after a bismuth meal was to the effect that the stomach was in a condition of ptosis and slightly dilated, with a one-fourth six hour residue. There was a doubtful stoma posterior to the stomach, close to the lesser curvature of the ptosed viscus, with some emptying through it. Dr. Wilkinson said that the main question for consideration was the advisability of opening the abdomen again and enlarging the gastro-enterostomy opening.

In the discussion which ensued, Mr. Syme said that at the original operation he had performed a posterior no-loop gastro-enterostomy, but it was quite possible that at that period a full 7.5 cm. opening was not made, as was the routine at present. He could not recollect the exact details as to the size of the stoma, but if it had closed, it was his first experience of closure of the stoma. The X-ray evidence tended to show that at all events the closure was not complete. It was possible that a peptic ulcer had originated in the vicinity of the opening and by cicatrization had led to some narrowing. It was also possible that malignant disease had supervened on the original ulcer, but he thought that neither of these contingencies was very probable. It appeared as if the stoma had not been large enough originally. Mr. Syme was of opinion that a further operation should be carried out. In the original operation no attempt had been made to close the pylorus.

Mr. Kilvington recollected one instance of posterior gastro-enterostomy in which closure had occurred and in which he had found it extremely difficult to do anything in the way of amelioration in the old operation area, on account of the dense adhesions. In that case he had resorted to an anterior gastro-enterostomy.

Mr. Alan Newton said that in 1915 he had collected all the cases for the previous ten years of operation for perforated duodenal ulcer at the Melbourne Hospital, in order to compare the after results of simple infolding of the ulcer with those of infolding combined with gastro-enterostomy. All the patients were given bismuth meals and subjected to X-ray examination. Speaking from memory, Mr. Newton stated that of approximately 20 examples of simple infolding, one patient manifested evidence of obstruction. Of the cases of infolding and gastro-enterostomy, 25% showed no efflux through the stoma; the presumption was that the stoma had closed and that the pylorus, in spite of infolding, was acting well. Experimentally, Mr. Newton had infolded the pylorus in dogs to an extent sufficient to narrow the lumen by about three-fourths, but subsequent X-ray examination failed to afford evidence of obstruction.

Dr. J. F. Wilkinson, in reply, said that in the patient under discussion there had been a pyloric ulcer, with definite narrowing of the lumen, at the time of the original operation. He considered that a large amount of apparent obstruction was due to spasm. On this account it was necessary to discount, to some extent, the X-ray findings of obstruction. In this particular case, Dr. Wilkinson thought that if the man could be kept under close observation and fed carefully, he could be enabled to get along in comfort. The difficulty was that the patient's livelihood obliged him to go back to farm life, with probably a rough diet. Perhaps the best result would be attained by enlarging the gastro-enterostomy opening.

Dr. Wilkinson's second patient was a young woman, aged 24, the subject of extreme obesity. She had always been stout, but in the last two years had become much more so. Her weight had reached 170 kilograms. Until two years ago she had menstruated normally, but for the last 24 months had had amenorrhœa. As a sugar tolerance test, she had been given a breakfast of 70 grm. of dextrose and one egg, with the result that after two hours the urine was loaded with sugar. Her blood sugar had been estimated as 0.125%. A skiagram of the skull showed a shallow *sella turcica*, closed in by bone. The optic discs were found to be somewhat pallid in the outer halves, but did not exhibit any evidence of disease.

Dr. Wm. Ostermeyer showed a man, aged 44, whom he regarded as the subject of *dementia paralytica*. He was admitted to hospital in a state of semi-coma. He was said to have had a fit just before admission. He had been in hospital with fits on two previous occasions. At the time of admission for the third time he lay curled up on his left side, with strong conjugate deviation of the head and eyes to the left. He resisted all attempts to move him. At a later examination, the left pupil was seen to be the larger; it was irregular and did not react to light. The right pupil reacted sluggishly, if at all. Fibrillary tremors were observed in the tongue, the articulatory movements of the lips were defective and the man's talk was gibberish. It was subsequently learned that the address he had given was a wrong one, as was also the name. Dr. Ostermeyer exhibited a specimen of the patient's handwriting, which was the typical rambling scrawl of the paralytic dement. His serum gave a positive Wassermann reaction, but the cerebro-spinal fluid showed neither excess of globulin nor cells. The chief point of interest about this man was the complete nature of the remission. Whereas before he had been obviously certifiable, he was now lucid and of apparently normal mentality.

Mr. Basil Kilvington presented a woman, aged 63, who had been admitted to the Melbourne Hospital on June 7, 1919, with a painful swelling on the inner aspect of the lower third of the left tibia, of four weeks' duration. There was no history of trauma. At first the swelling was soft, but it became rapidly larger and harder. The Wassermann and von Pirquet tests did not give rise to reactions. Mr. Syme had operated and shelled out the tumour by blunt dissection. According to a note made at the time, the growth had no macroscopical connexion with the tibia. The pathological report on this specimen was that it was a sarcomatous growth of mixed-celled type. The patient was discharged on July 20, 1919, but was readmitted one week later with fresh swelling and puffiiness at the site of the operation. The swelling was fluctuant in some areas and hard in others. Subsequently Mr. Zwar performed Farabœuf's amputation. A second pathological report of periosteal sarcoma of tibia was received on August 7, 1919. On April 28, 1920, this woman came back to hospital on account of a swelling on the middle of the anterior margin of the right tibia, which she stated first appeared three weeks previously and was rapidly growing. The swelling was painful, tender and firm to palpation.

Mr. Kilvington removed a piece for examination and received a report that the specimen was of endotheliomatous or sarcomatous nature and non-inflammatory. An X-ray screen examination of the chest revealed nothing abnormal. On June 6, 1920, the right palpebral fissure was observed to be narrower than the left. Examination disclosed a swelling at the outer margin of the orbit. Since that date the swelling had increased in size. A skiagram of the orbit showed some absorption of bone and loss of detail. Mr. Kilvington said that the case raised the interesting question as to whether the neoplasm which appeared in the right tibia was a metastatic deposit following that in the left, or whether the two growths were each primary and independent of one another. He was now treating this patient with Coley's fluid. The fourth injection of 0.47 c.cm. had caused only a slight febrile reaction.

Mr. Alan Newton showed an example of carcinoma of the tongue in a woman, aged 67. There had been soreness of the tongue for 12 months and a lump on the edge of the tongue appeared four months previously. There had been no pain, but smarting when hot fluids were taken, had been troublesome. Her speech was very indistinct. On examination, a condition of superficial glossitis was evident. A papular growth with indurated base was situated on the side of the tongue, near the tip. The sub-mental glands were palpable. A positive Wassermann reaction had been obtained. It was interesting to note that this woman had had a growth removed from the lip eight years before.

Dr. S. W. Patterson exhibited a number of pathological specimens of various tuberculous pulmonary lesions.

Naval and Military.

APPOINTMENTS.

The following appointments, etc., have been notified in the *Commonwealth of Australia Gazette*, No. 61, of July 22, 1920:—

Australian Imperial Force.

First Military District.

Captain P. A. Earnshaw, Australian Army Medical

Corps, having resigned, his appointment in the Australian Imperial Force is terminated in England on 29th April, 1920, but to take effect from 29th May, 1920.

Second Military District.

Captain (temporary Major) W. R. Young, Australian Army Medical Corps, having resigned, his appointment in the Australian Imperial Force is terminated in England on 3rd May, 1920, but to take effect from 17th June, 1920.

Third Military District.

Captain W. I. Hayes, Australian Army Medical Corps, having resigned, his appointment in the Australian Imperial Force is terminated in England on 3rd May, 1920, but to take effect from 26th June, 1920.

Captain H. C. Trumble, Australian Army Medical Corps, having resigned, his appointment in the Australian Imperial Force is terminated in England on 23rd April, 1920, but to take effect from 7th June, 1920.

Fourth Military District.

Captain H. W. H. Cairns, Australian Army Medical Corps, having resigned, his appointment in the Australian Imperial Force is terminated in England on 1st May, 1920, but to take effect from 31st May, 1920.

Fifth Military District.

Major J. B. Lewis, Australian Army Medical Corps, having resigned, his appointment in the Australian Imperial Force is terminated in England on 8th May, 1920.

To be Captain—

Captain, provisionally, B. D. Gibson, Australian Army Medical Corps, 26th October, 1915.

APPOINTMENTS TERMINATED.

First Military District.

Major J. MacDonald, O.B.E., 7th July, 1920.

Third Military District.

Major J. P. Kelly, 24th August, 1920.

Fourth Military District.

Lieutenant-Colonel L. W. Jeffries, O.B.E., D.S.O., 13th July, 1920.

Australian Military Forces.

APPOINTMENTS, PROMOTIONS, ETC.

First Military District.

Australian Army Medical Corps—

Captain E. C. G. Page to be transferred to the Unattached List, 1st June, 1920.

Second Military District.

Australian Army Medical Corps—

Captain and Brevet-Major H. L. St. V. Welch, D.S.O., to be transferred to the Reserve of Officers, 1st Military District, and to be Lieutenant-Colonel, 1st May, 1920.

Australian Army Medical Corps Reserve—

Honorary Major M. O'G. Hughes to be Honorary Lieutenant-Colonel, 1st July, 1920.

Honorary Captain F. C. Thompson to be transferred to the Reserve of Officers, 5th Military District, and to be Captain, 1st March, 1920.

Third Military District.

Australian Army Medical Corps—

The resignation of Captain J. L. Jona of his commission is accepted, 18th November, 1919.

Australian Army Medical Corps Reserve—

Honorary Captains C. T. Stephen and C. P. W. Dyring to be granted the temporary rank and pay of Majors whilst employed at No. 11 Australian General Hospital, dated 1st March, 1920, and 1st June, 1920, respectively.

Fourth Military District.

Australian Army Medical Corps Reserve—

To be Honorary Major, Honorary Captain H. F. Shorney, 1st May, 1920.

The resignation of Honorary Captain O. M. Moulden of his commission is accepted, 30th June, 1920.

Fifth Military District.

Australian Army Medical Corps Reserve—

Honorary Captain A. Juett to relinquish the temporary rank of Major and to be granted the temporary rank and pay of Lieutenant-Colonel whilst employed at No. 8 Australian General Hospital, 24th May, 1920.

Honorary Captain W. J. Patterson to be transferred from the Australian Army Medical Corps Reserve 6th Military District, 1st June, 1920.

Sixth Military District.

Australian Army Medical Corps Reserve—

Honorary Captain W. J. Patterson to be transferred to the Australian Army Medical Corps, Reserve, 5th Military District, 1st June, 1920.

The following appointments have been notified in the *Commonwealth of Australia Gazette*, No. 63, of July 29, 1920:

Second Military District.

To be Captains—

H. G. Leahy, M.C., H. Dolman.

Third Military District.

To be Captains—

H. Mendelsohn, M.C., R. S. Andrews.

Readers of the *Journal* are informed that when they desire to express in the correspondence columns an opinion on questions of policy which are at that time under consideration by any Branch of the British Medical Association in Australia, their contributions must be signed. The practice of writing letters over an assumed name or initials has become unnecessarily common. At times, no doubt, there is a valid reason for anonymity. It should, however, be remembered that in many cases the arguments put forward lose considerably in effectiveness by the fact that personal influence is excluded.

PUBLIC HEALTH OF NEW SOUTH WALES.

(Continued from Page 21.)

Private Hospitals.

Dr. F. M. Suckling publishes a special report on the operation of the *Private Hospitals Act, 1908*. During the course of the twelve months, 432 private hospitals in the metropolis and 77 in country towns were re-inspected. It appears that there are 395 private hospitals with duly qualified resident managers. In addition, there are 143 with uncertified resident managers. These institutions are relicensed as long as they are satisfactorily conducted. In small, remote country towns some of the institutions are registered because they are the only hospitals in the district. During the year ten new licences were granted to uncertified applicants. In each case the licences were recommended by two medical practitioners and the premises were carefully inspected. A difficulty appears to arise in hospitals with uncertified resident managers. In these places there is frequently a change of the approved certificated general nurse and the licensee frequently neglects to notify the Department when the approved nurse leaves. At times, the hospital is conducted without a certificated nurse. Dr. Suckling thinks that it would be advisable if licences were granted only to nurses holding the certificates of the Australasian Trained Nurses' Association. It was found on the second inspection of unsatisfactory hospitals that the defects in the majority of instances were being remedied.

Sanitary Inspection.

The Chief Sanitary Inspector, Mr. E. A. Cresswick, deals with the work carried out by his staff during the course of the year. He has associated with him three sanitary inspectors. Systematic inspection was carried out of nine country towns and full reports were submitted. In these reports the administration of sanitary matters, the water supply, the disposal of sewage and liquid wastes, the collection and disposal of night-soil and garbage, the supplies of meat and food, noxious trades, cemeteries, hotels, infectious diseases, disinfection and vital statistics are dealt with. The Chief Inspector, however, does not even mention the nine country towns by name, nor is any intimation concerning these interesting details given in his report.

Visits were paid to 37 country towns for the purpose of ascertaining whether sanitary defects discovered at a previous inspection had been remedied. In two instances the local authorities had failed to carry out the work required. The Crown Law Department was therefore requested to take legal action. Notices were served on two councils to abate serious nuisances, while several local authorities were warned that legal action would be taken, unless the recommendations of the Board were carried into effect.

It is stated that in five instances outbreaks of infectious disease were investigated by the sanitary inspectors. We have called attention to the anomaly of such an action. A sanitary inspector is certainly not qualified to trace the cause of an outbreak of an infective process nor to deal with any nidus of infection.

Mr. Cresswick points out that many of the councils do not employ a qualified sanitary inspector. As a result, only 71 local officers are authorized to recommend the closing of insanitary buildings, under the provisions of the *Public Health (Amendment) Act, 1915*. The remainder of his report deals with the number of inspections of wharves, ships, hotels, septic tanks, water supplies, etc., etc.. In regard to noxious trades, he points out that 605 persons hold licences and that some of the licensees hold two or more licences. In the majority of instances the supervision exercised by the local authority was found to be good. In three instances it is stated to have been "poor," which we presume means bad. Prosecutions for breaches of the *Noxious Trades Act* were instituted on 21 occasions. The fines and costs varied between £1 and £10 6s..

Local Health Administration.

Part II. of the general report consists of three reports, the first by Dr. J. S. Purdy, D.S.O., the Medical Officer of Health of the Metropolitan Combined District, the second by Dr. Robert Dick, Medical Officer of Health of the Hunter River Combined District, and the third by Dr. J. F. Bartley, Medical Officer of Health for the Municipality of Broken Hill. Much of the information contained in these reports is necessarily included in the statistical returns of the main report.

(To be continued.)

Correspondence.

PULLING TOGETHER.

Sir: In *The Medical Journal of Australia* of June 19 last, I read your leading article, "Pulling Together," with the greatest pleasure.

When retracing my steps during the long wandering reveries of many years, the following passage in the above article—"How many are full of gratitude when their relatives are ill and the doctor's services are required; when the patient has recovered and the doctor has to be paid, the feeling of indebtedness is forgotten"—brought very forcibly to my mind a little verse, which I heard many years ago by an old medical friend in England, and which still holds good. This has remained fixed in my memory through experience and sings eloquently of the same theme. Here it is—

"God and the doctor men adore,
When sickness comes but not before;
When all is well and everything righted,
God is forsaken and the doctor slighted."

My excuses for sending this to you are:—

(*a*) The verse's close analogy to your able article; and

(*b*) Not having heard it among my medical friends in Australia.

Yours, etc.,
MORGAN RICHARDS, M.D..

"Bryn Hyfryd,"
Kalamunda, Western Australia,
July 5, 1920.

LIGATURE OF THE UTERINE ARTERIES.

Sir: There appears to be some doubt as to who was the first to improve on the old method of performing hysteromyomectomy by that of ligaturing separately the uterine and ovarian arteries and converting a very serious operation into one of the simplest in surgery.

Professor Watson undoubtedly deserves the credit for being the first one to demonstrate this method in Australia, the secret of which was to prevent the uterine artery pumping its blood into the tumour while the surgeon was rabbiting about the pelvis, which used to be the case prior to this method and, as well as producing an almost bloodless field, saved the patient the blood originally in the tumour.

It was in 1898 that Professor Watson first demonstrated this method in Melbourne shortly after he had done so in Adelaide and Sydney.

I well remember when Howard Kelly's great work on gynæcology, containing a description of the operation, was published, chaffing the Professor with the fact that he had seen the proofs before they were put into the printer's hands.

In July, 1898, Professor Watson showed me the method on a multiple myomata impacted in the pelvis. I surprised myself by leaving the infundibulo-pelvic fold on the right side till the whole tumour was out of the abdomen and by seeing the patient leave the operating table with a pulse of 60 and no loss of blood.

The operation performed prior to 1898 was that described by Baldy, of Philadelphia, in which the broad ligament was secured in pieces from above down and masses of tissue tied with nothing in them except occasionally the ureter.

One leading Melbourne gynæcologist in 1898 still practised the method of securing the cervical stump with silver wire in the lower end of the abdominal wound.

The mode of treating the pedicle of an ovariotomy at this period was to transfix the pedicle in the centre and tie off in two pieces. The outer ligature had a bad habit of slipping and letting go the ovarian artery. To remedy this, the late Dr. O'Sullivan was accustomed to use what he termed a supplementary ligature through the edge of the infundibulo-pelvic fold, thus secured the ovarian artery further out.

A *post mortem* in one case demonstrated the value of this ligature, the outer loop having slipped, but not a drop of blood was lost.

Resident surgeons at the Women's and St. Vincent's Hospitals will remember the Keith glass drainage tube being placed in the lower end of the abdominal wound for all hysterectomy and pelvic operations; also the great amount of shock which followed these operations and one gave a sigh of relief when at the end of 48 hours there was no sign of a slipping ligature or a tied ureter and the sister in charge reported a successful bowel action.

Yours, etc.,
G. ALFRED HAGENAUER.

Sale,
July 8, 1920.

A HUMAN TAPEWORM NEW FOR AUSTRALIA.

Sir: I wish to record the discovery of a tapeworm, *Hymenolepis diminuta*, as a parasite in an infant of 13 months.

This tapeworm, so far as I can ascertain, has not previously been recorded from a human in Australia, though it occurs in rare instances in Europe and America.

I am at present investigating the infection with this tapeworm of rats caught in the locality which is of interest

on account of the possibility of direct development of the parasite and hope shortly to publish a full account.

Yours, etc.,

CHARLES BADHAM.

Kendall, North Coast, New South Wales,
July 15, 1920.

THE ACTION OF NOV-ARSENO-BILLON.

Sir: I have read with interest recent letters dealing with marked reaction supervening on the intravenous administration of nov-arseno-bilion. In the early days of its employment at the London Hospital cases occasionally occurred as a result of which Dr. Sequeira and his *confrères* introduced the following simple routine:—

(*a*) Preparation by purgation of the patient.
(*b*) Abstention for several hours from a meal.
(*c*) Urine tests in each case; anything over a trace of albumin contra-indicating.
(*d*) Freshly distilled water; and
(*e*) Immediate solution and administration of the drug.

During fifteen months in which I was engaged on venereal and genito-urinary work at the hospital, I do not recollect ever seeing a single case of marked reaction, and the clinic is the largest probably in England. During six months' attendance at the Female Lock Hospital, where substantially the same routine prevails I understand, I cannot recall observing any instances either. I applied the same rules in my private practice and occasionally on emergency utilized plain, thoroughly-boiled main water with similar results.

The rule as to a meal is, I believe, of importance. The real danger in the use of "914" is undoubtedly failure to inject fully into the vein, filtration into the tissues often producing serious results.

I recollect seeing several very bad arms come up to various hospitals in London as the result of unskilful attempts outside the hospitals. One man I remember was incapacitated from work for about four months.

So insistent was Dr. Sequeira on the necessity for experience in intravenous work that he never permitted practitioners who attended from time to time for a course of several months' study, to administer "914" except under the direct supervision of one or other of his assistants. He then generally insisted on the employment of the pressure apparatus used at the hospital (whereby a dose was diluted with about five ounces of distilled water) for a number of times before he permitted practice in concentrated solutions, always under the supervision, however, until about fifty doses had been successfully administered.

As regards dosage, a less dose than 0.6 gramme was never administered to adults at the hospital and I am inclined to think minimal doses would not obviate reaction, if the other precautions were not adopted.

Yours, etc.,

T. S. GREENAWAY.

110 Collins Street, Melbourne,
July 21, 1920.

PAYING HOSPITALS.

Sir: None can dispute the abuse of the system of charity carried out at our public charitable institutions. The latest source of revenue, I note, is from the venereal diseases branch at "The Melbourne," where an income from those attending is expected to reach close upon £2,000 a year. "Medical Staff," "Medical Staff No. 1" and "Medicinus" have set out only what is known to everyone, but the whole matters rests entirely in the hands of the men complaining. Their letters speak in self-condemnation. Why do they and others do the work complained of? No other members of the profession desire that they should do what they know full well is wrong to the suffering poor, wrong to their profession and wrong to themselves. Why should not the honorary medical and surgical staffs, or the British Medical Association for them, dictate the conditions under which their gratuitous services are available?

For the information of all whom it may concern, I take this opportunity of directing attention to the *British Medical Journal*, May 29 (page 752), under the heading: "Payment of Visiting Staff of Voluntary Hospitals." In the report of a conference of representatives of the honorary visiting staffs of some of the larger voluntary hospitals of the midlands, which was held on May 18 at Leicester Royal Infirmary, many hospitals were represented, including the Birmingham, Burton, Coventry, Derby, Leicester, Loughborough, Manchester, Northampton, Nottingham, Salford, Sheffield, Stoke-on-Trent, West Bromwich and Wolverhampton and thirty-six representatives were present, the chair being taken by Sir Richard Luce, of Derby. The questions brought up for discussion were:

(*i.*) The desirability of payment for the work done at their hospitals on behalf of the Ministry of Pensions, the Ministry of Health or the local medical health authorities, and, if so, at what rates?

(*ii.*) Whether the time has come to ask for payment of the visiting staff for all work done by them at the hospitals and, if so, whether this should take the form of an honorarium, a salary, or a percentage of the expenses of the hospital, etc., etc..

After full discussion the following resolutions were carried:

(*i.*) That payment should be made, etc..

(*ii.*) That the time has not come for honorary visiting staffs to ask for payment for work done at hospitals whose incomes were derived from voluntary contributions, etc..

In dealing, therefore, with the charges brought forward by those who are aggrieved, there is but one thing to do. Refuse to do the work for those who cannot claim to come under the heading of "poor and needy."

Yours, etc.,

RICHARD JONES.

110 Collins Street, Melbourne.

Proceedings of the Australian Medical Boards.

NEW SOUTH WALES.

The undermentioned have been registered under the provisions of the *Medical Act, 1912 and 1915*, as duly qualified medical practitioners:—

Austin, Robert Blackie, M.B., Bac. Surg., 1910 (Univ. Glasgow), Neutral Bay.
Boyt, Mavis Amelia Eleanor, M.B., Bac. Surg., 1920 (Univ. Melbourne), Broken Hill Hospital.
Cook, Leonard Roy, M.B., Bac. Surg., 1913 (Univ. Melbourne), Hay.
Coutts, Donald Dunbar, M.B., Bac. Surg., 1914 (Univ. Melbourne), Nyah West, Victoria.
Ferris, George Thomas, M.B., Mast. Surg., 1920 (Univ. Sydney), Redan Street, Mosman.
Furner, Carl Russell, M.B., 1920 (Univ. Sydney), Balmain Hospital.
Johnson, Hubert Edwyn, M.B., Bac. Surg., 1908 (Univ. Edin.), Hay.
Lang, Alexander Allison, M.B., Bac. Surg., 1916 (Univ. Melbourne), Willoughby.
McLaughlin, Arthur Ivan Granville, M.B., Mast. Surg., 1920, (Univ. Sydney), Sydney Hospital.
Paling, John Marie Anthony, M.B., Mast. Surg., 1920 (Univ. Sydney), Royal Prince Alfred Hospital.
Russell-Jones, Roy Warren, M.B., Bac. Surg., 1913 (Univ. Edin.), Woollahra Point.

Applications for registration of additional qualifications:—
Burfitt, Mary Boyd, Mast. Surg., 1910 (Univ. Sydney).
May, Leonard, F.R.C.S. (Univ. Edin.), 1919.

VICTORIA.

The undermentioned have been registered under the provisions of Part I. of the *Medical Act, 1915*, as duly qualified medical practitioners:—

Brown, Charles James Officer, M.B., B.S., Melb., 1920, "Wiltara," Mercer Road, Malvern.
Derrick, Edward Holbrook, M.B., B.S., Melb., 1920, Melbourne Hospital.
Hobbs, Jack Rawlinson, M.B., B.S., Melb., 1920, Geelong Hospital.
Lee, Alan Edward, M.B., B.S., Melb., 1920, Ballíang.

Mortensen, Henry Newman, M.B., B.S., Melb., 1920, St. Vincent's Hospital, Melbourne.
Mackay, Eric Eccles, M.B., B.S., Melb., 1920, Box 37, Broken Hill, New South Wales.
Niall, Francis John, M.B., B.S., Melb., 1920, Melbourne Hospital.
Pearse, Thomas Sargent, L.R.C.P. *et* S., Edin., L.F.P.S., Glas., 1904, Railway Hotel, Albert Street, Brunswick.
Prendergast, Edward Joseph, M.B., B.S., Melb., 1920, 41 Marine Parade, St. Kilda.
Stewart, Alexander, M.B. *et* C.M., Glas., 1895, c/o. Mr. Penwarden, Mayston Street, Upper Hawthorn.
Telford, Robert Whitson, M.B. *et* C.M., 1905, M.D., 1909, Edin., D.P.H., Camb., 1912, c/o. Mr. Morell, 480 Punt Road South Yarra.

Additional diplomas registered:—
Stewart Osburn Cowen, M.D., Melb., 1920.
Thomas Ernest Victor Hurley, F.R.C.S., Eng., 1919.

Names of deceased practitioners removed from the Register:—
George Ernest Morrison.
Wilberforce Vaughan Eaves.

Medical Appointments.

Dr. R. L. Rosenfield (B.M.A.) has been appointed Honorary Aural Surgeon to the Victorian Eye and Ear Hospital, the position having been rendered vacant through the retirement of Dr. R. E. Shuter (B.M.A.).

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, locum tenentes sought, etc., see "Advertiser," page xxvii.

University of Sydney: Chair of Surgery.
University of Sydney: Chair of Physiology.
Hamilton Hospital and Benevolent Asylum, Victoria: Medical Officer.

Medical Appointments.

IMPORTANT NOTICE.

Medical practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, 429 Strand, London, W.C.

Branch.	APPOINTMENTS.
VICTORIA. (Hon. Sec., Medical Society Hall, East Melbourne.)	All Friendly Society Lodges (other than the Grand United Order of Oddfellows and the Melbourne Tramways Mutual Benefit Society), Institutes, Medical Dispensaries and other Contract Practice. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association.
QUEENSLAND. (Hon. Sec., B.M.A. Building, Adelaide Street, Brisbane.)	Australian Natives' Association. Brisbane United Friendly Society Institute. Cloncurry Hospital. Stannary Hills Hospital.
SOUTH AUSTRALIA. (Hon. Sec., 3 North Terrace, Adelaide.)	Contract Practice Appointments at Renmark. Contract Practice Appointments in South Australia.
WESTERN AUSTRALIA. (Hon. Sec., 6 Bank of New South Wales Chambers, St. George's Terrace, Perth.)	All Contract Practice Appointments in Western Australia.
NEW SOUTH WALES. (Hon. Sec., 30.-34 Elizabeth Street, Sydney.)	Australian Natives' Association. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham Dispensary. Manchester Unity Oddfellows' Medical Institute, Elizabeth Street, Sydney. Marrickville United Friendly Societies' Dispensary. North Sydney United Friendly Societies. People's Prudential Benefit Society. Phœnix Mutual Provident Society.
NEW ZEALAND: WELLINGTON DIVISION. (Hon. Sec., Wellington.)	Friendly Society Lodges, Wellington, New Zealand.

Diary for the Month.

Aug. 9.—N.S.W. Branch, B.M.A., Ethics Committee.
Aug. 10.—Tas. Branch, B.M.A..
Aug. 12.—Vic. Branch, B.M.A., Council.
Aug. 13.—N.S.W. Branch, B.M.A., Clinical.
Aug. 13.—S. Aust. Branch, B.M.A., Council.
Aug. 17.—N.S.W. Branch, B.M.A., Executive and Finance Committee.
Aug. 17.—Illawarra Suburbs Med. Assoc. (N.S.W.).
Aug. 18.—W. Aust. Branch, B.M.A..
Aug. 18.—North-Eastern Med. Assoc. (N.S.W.).
Aug. 19.—Western Suburbs Med. Assoc. (N.S.W.).
Aug. 24.—N.S.W. Branch, B.M.A., Medical Politics Committee; Organization and Science Committee.
Aug 25.—Vic. Branch, B.M.A., Council.
Aug. 26.—S. Aust., B.M.A..
Aug. 27.—N.S.W. Branch, B.M.A..
Sept. 1.—Vic. Branch, B.M.A..

EDITORIAL NOTICES.

Manuscripts forwarded to the office of this journal cannot under any circumstances be returned.

Original articles forwarded for publication are understood to be offered to *The Medical Journal of Australia* alone, unless the contrary be stated.

All communications should be addressed to "The Editor," *The Medical Journal of Australia*, B.M.A. Building, 30-34 Elizabeth Street, Sydney. (Telephone: City 2645.)

We are seeking copies of our issues of February 14 and 28, 1920. There has been an unusual demand on our stock, which is now depleted. Members who do not keep *The Medical Journal of Australia* for binding, are requested to send us their copies, on receipt of which the usual payment of the face value will be made.